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THE TEST-DIET
IN
INTESTINAL DISEASES

PROF. DR. ADOLF SCHMIDT

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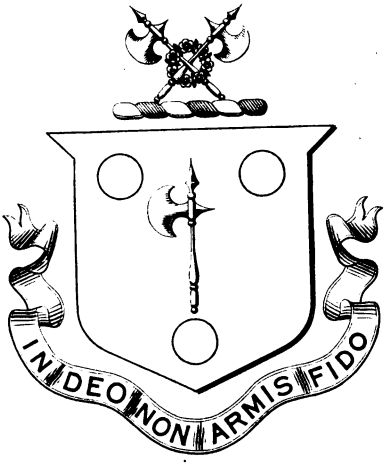
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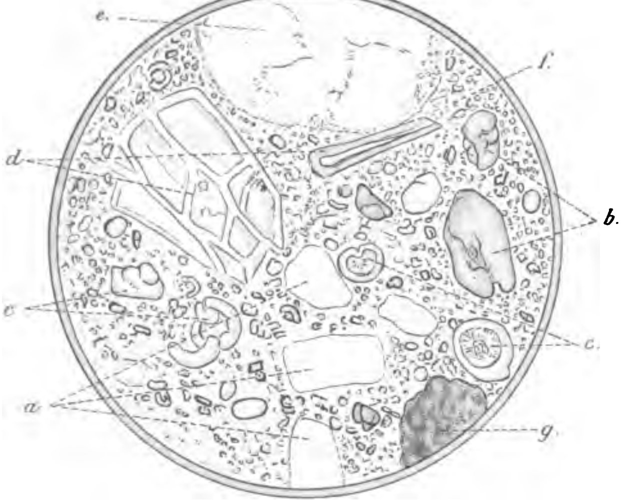
John Lovett Morse

Fig. I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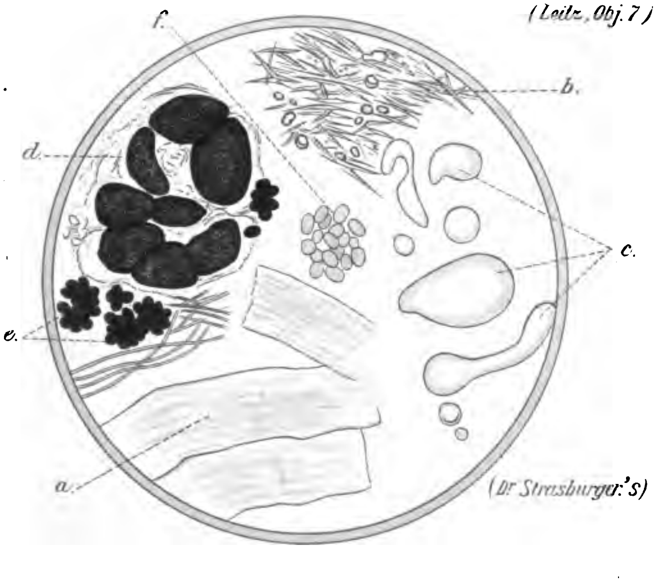
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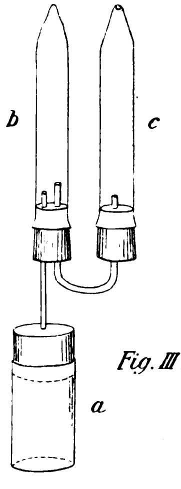

Fig. III.

(Dr Strasburger's)

FIG I. NORMAL TEST-DIET FECES (combined picture). (Leitz, Obj. 7.)

a, Muscle fibre remains.

b, Yellow calcium salts.

c, Soaps.

d, Chaffy remains.

e, Empty potato cells.

f, Detritus.

g, Cocoa remains.

FIG. II. PATHOLOGIC INGREDIENTS IN THE TEST-DIET FECES (combined picture). (Leitz, Obj. 7.)

a, Large muscle fragments.

b, Fatty acids and soap needles.

c, Neutral fat.

d, Starch granules in potato cells.

e, Granulose spherical and thread fungi.

f, Yeast cells.

FIG. III. FERMENTATION TUBES (after Dr. Strasburger).

THE EXAMINATION OF THE FUNCTION OF THE INTESTINES

BY MEANS OF

THE TEST-DIET

ITS

APPLICATION IN MEDICAL PRACTICE AND ITS DIAGNOSTIC AND THERAPEUTIC VALUE

BY

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AUTHORIZED TRANSLATION FROM THE LATEST GERMAN EDITION

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PREFACE.

THE following pages owe their origin to a series of lectures, which I delivered in the summer of 1903 at the instance of my colleagues. They sum up for medical purposes the results of my continued investigations and observations for about eight years. The desire to obtain an examination for the function of the intestines, *that could be carried out in practice,* analogous to the usual examinations of the stomach contents, and to advance by means of it the diagnosis and therapeutics of the diffuse intestinal diseases, in the same way as it has already been accomplished in the pathology of the stomach, was the incentive which led me on from the beginning. The great deviations, which my investigations and those of my co-worker, Dr. *Strasburger,* had to make in order to secure a scientifically established basis for our practical methods, have made it impossible to put this goal in the foreground, as I had it in mind to do. For this reason and partly also because our works were scattered in the special literature, our methods for examining the function of the intestine have not yet been able to secure a firm footing in practice. To many they at first seemed too complicated, a fault, which, I hope, has been almost altogether removed. The conviction, that our functional examination, though still having many gaps, can be of service to the practitioner in many respects, even in its present form, and the encouraging approbation of colleagues, have been to me the cause for this comprehensive presentation. At the same time it affords me a desired opportunity for the publication of some recently obtained results.

ADOLF SCHMIDT.

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I. THE EXAMINATION OF THE FUNCTION OF THE INTESTINE.

Functional Diagnosis of the Stomach and of the Intestine.—Experiments in Digestion.—Conditions of a Rational Test of the Function of the Intestine.—The Examination of the Content of the Stomach as Indicator.

To ANY one who compares the present state of intestinal pathology with that of the pathology of the stomach, there can be no doubt, that in spite of the various advances we have made, especially in the field of local inflammations (appendicitis) of stricture and of obstruction of the intestine, the former has nevertheless remained very far behind the latter. This is especially true for those diffuse diseases of the intestine and its adnexa (the liver and the pancreas) which have no broadly recognizable symptoms. These will form the subject of our consideration primarily; in other words, the lighter grades of inflammation and the numerous other so-called functional diseases, for which it has hitherto proved impossible to find an anatomic substratum. In the pathology of the stomach it is in the knowledge of just *these* forms of diseases that extraordinary progress has been made in the last two decades, and this we owe to the use of the stomach-tube for the purposes of diagnosis *(v. Leube)* and to the introduction of the test-breakfast and the test-meal *(Ewald, Riegel);* in short, to the creation of a functional diagnosis of the diseases of the stomach.

The aim of *functional diagnosis,* which is now being striven for in all branches of internal medicine, is the recognition of disturbances in the physiologic process (the functions) of organs; even then when objective signs of the disturbances are still wanting, when perhaps only indefinite subjective sensations are present or even no symptoms at all, as happens, for example, in *achylia gastrica.* In order to obtain this, an arbitrarily chosen task is imposed on the particular organ and from the way it discharges this task, its efficiency is inferred. The difficulties in functional diagnosis, which we need absolutely owing to the natural limitation of our anatomic knowledge and of our sense-perceptions, lie in finding suitable criteria that can be introduced into general practice, for testing the general operation or the total work, which is divided up according to the various functions of organs, and in the demarcation of the ranges of physiologic fluctuation in their operation. The latter must naturally be accurately known before we can infer to pathologic conditions. Such a criterion once obtained, we could by means of it advance our diagnostic and therefore our therapeutic knowledge considerably. This is proved by the functional examination of the eye by means of the altogether arbitrary concept of visual acuity; this is proved for internal medicine by the advances in the pathology of the stomach. As a result of investigations with the stomach-tube, we are now in a position to control the work of the stomach in its finest details, and to test separately the part played in it by its three chief functions, motility, secretion, and absorption.

In intestinal pathology we are still far from this ideal. Owing to the inaccessibility of this organ the introduction of tubes and similar instruments to aid in palpation and illumination of the mucous membrane, and the insufflation of air and liquid, from above, are out of question. Such methods can be employed only from the anus and may give us our bearings as to the condition of the rectum, but less frequently as to the upper sections of the colon. *Hemmeter* (1) and *Kuhn* (2) have repeatedly passed the pylorus by way of the stomach, but there can be no question of the general application of their methods. Similarly the obtaining of duodenal secretion from the empty stomach, to which *Boas* (3) first called attention, is connected with special preliminary conditions, which are found only exceptionally. Disregarding the aids of physical diagnosis, of inspection, palpation, percussion, and auscultation of the abdomen, and of the evidences which are furnished by the examination of the urine and of the other organs in general, we are essentially dependent upon the examination of the feces. But with the examination of the feces peculiar circumstances are connected. In practice it is used entirely too seldom and it is valued too little. I believe I offend no one by asserting that, aside from hospital physicians and specialists on the diseases of the stomach and intestines, scarcely any practitioner proceeds with the investigation of the feces differently than the layman himself: *i.e.*, he inspects it and smells at it, and, at most, stirs it with a stick. How does this happen? Is the prudery of the public and the repulsiveness of the examination of the feces the cause, or does the analysis of the feces in reality promise such slight results as not to be worth the trouble? I believe that both points enter here; for since *Nothnagel's* investigations, dating from 1884 (4)—the valuable results of which have unfortunately found admission only in a limited circle of physicians—to the very present time, the clinical analysis of feces has made scarcely any progress. It cannot be disputed that the method of examining the feces used at present only rarely gives results useful for diagnosis.

Still we must not forget the great advances which we owe to the numerous *experiments in metabolism* or *digestion* performed on normal and pathologic intestines. The experiment in digestion is the given criterion of the examination of the function of the intestine and its adnexa; it shows us how much and what elements of the food leave the body undigested. We owe to the experiment in digestion practically everything that we know of the functional disturbances of the liver and the pancreas and in regard to their secretion. It has also proved itself valuable for the recognition and differentiation of various intestinal disturbances; but (and that is the difficulty) it requires apparatus too complicated ever to become the property of all physicians; its application will always be confined to clinics and large hospitals.

Experiments in digestion have also other defects, which are not to be passed over in silence, when we are concerned with the prerequisities of a functional test suitable for clinics (5). In the first place, the choice and composition of the nutriment is of decided importance for the amount of the food-remains that appear in the feces. In previous experiments, however, the choice of diet was in many respects made without regard to the efficiency of the intestine; at any rate there was never any question of uniformity in the diet—an absolute requirement for comparative investigations. Then through the usual chemical methods for determining the nitrogen and fat in the feces, the remains of albumin and fat from the food which go to waste has not been indicated with sufficient accuracy. They were always measured together with the substances containing nitrogen, with the fat-bodies excreted from the body itself, and we have no possibility of separating these two altogether heterogenic groups from each other.

Now under certain circumstances, *e.g.*, in the case of a diet that contains little waste material, the excretions from the body constitute a very considerable portion of the content of nitrogen and fat in the feces. In a mixed diet, *e.g.*, the nitrogen that comes from the body amounts, according to *Rieder,* to about 29 per cent. of the total content of nitrogen in the feces (6), and *Strasburger* (7) has calculated, that with an easily digestible diet at least half of the entire nitrogen in the feces is to be credited to the fecal bacteria. As regards fat, *Rubner* (8) found 3 to 6 grams of ethereal extract in the daily feces, even with an almost fat-free diet. These figures prove that the results of the chemic analysis of the feces are quite unreliable. It appears that it will fail altogether where we are concerned with slight deviations from the normal and with the investigation of the finer disturbances of intestinal activity. To this is to be added the fact that up to very recently we did not have any method for determining the carbohydrate remains in the feces, that was practicable even to a slight degree. The method developed by *Strasburger* (9) in 1901 first proved sufficiently reliable and sensitive, but it is so difficult, as to demand the hand of an experienced chemist. A third chief defect of the chemical analysis of the feces consists in the fact that it does not give us any kind of information as to the form in which the undigested food-remains leave the body. A question that constantly forces itself upon us in the functional examination of the intestines is this: Are the undigested remains which are here evacuated with the feces only indigestible waste matter, or have nutritive elements, which ordinarily should have been digested, been evacuated at the same time. For the clinician it is not a matter of indifference whether the albumin-remains in the feces come from undigested muscle fibers or from connective tissue, and whether the undigested starch granules are free in the feces or are protected from the influence of the digestive juices by cellulose walls. Concerning these matters only the macroscopic and microscopic investigation can give us information, and it would certainly be perverse to desire to abandon these simple methods for the sake of chemic analysis.

The question suggests itself whether, retaining the principle, in itself correct, of the experiments in digestion, but with a simplified technic, and avoiding the sources of error here enumerated, it would not be possible to find a test of the function of the intestine that would be available for general practice. If this thought is followed out further, we have at once these points:—

1. *We must have as our starting point a uniform composition of the feces—a normal feces.* This normal feces need not, however, have so wide a scope as *Praussnitz* (10) would have it, so that it could come from any food at all, as long as it contained little waste matter, but it must always presuppose *one and the same altogether uniformly composed diet, the "test-diet."* Everything depends upon the proper selection and universal employment of the test-diet. Any one who has ever made investigations of feces knows that, so far as the amount and kind of the remains are concerned, the significant conditions found depend primarily on the amount and kind of food that has been served, and only secondarily on the conditions of the digestive organs. At least, I would regard it as fallacious, under all circumstances, to draw inferences with regard to the functioning of the digestive organs from the conditions of remains of food substances in the feces, without first knowing exactly what food has been taken in the last two days. Just in the changing composition of the diet lies the diffi-

culty of making the results of the investigation of the feces of value for practice. One need only examine his own stool macroscopically and microscopically regularly, for a few days, especially after dinner, or supper, and he will wonder at the variety of things that pass the intestinal canal undigested. To those interested in the variable forms of human excrement, the excellent book of *van Ledden-Hulsebosch* (11) gives the desired information.

2. *The stool investigation must be simplified as much as possible.* All methods which cannot be carried out in practice are more or less worthless. They may be of great service for scientific investigation, but they will never be used among physicians. We need only to study the examination of the content of the stomach and see what few tests we can depend upon. Of all the numerous methods for determining the acidity, only the phloroglucin test, the lactic acid test, and the total acidity test are used. Everything else has been left in the clinics. The same thing is true in the examination of the urine. Of the innumerable chemical tests only the simplest albumin and sugar tests have become the common property of physicians. Similarly for microscopy, as only the making of the simplest preparations and stains comes into consideration. It might appear strange that I should lay so much stress on simplicity in the methods of examining the feces, since the "fermentation test" and the "digestion test," which were first proposed by me, have been criticised as being too detailed. But as I have already pointed out, I have in the course of time learned to simplify my methods of investigation more and more. I am willing to admit that those tests in their original form were far too complicated, but I may perhaps urge in extenuation that only by the roundabout ways of the most exact investigation of details can results be obtained

for general utility. Now, after the extent and fluctuations of the hydrochloric acid secretion of the stomach has been determined through many examinations after the most difficult chemic methods have been employed one after the other, we are able to derive the proper inferences from even the simplest color-reactions. Such, too, will be the course of the investigation of the feces.

The examination of the stomach-content cannot be too closely kept in sight as an indicator. The method of functional examination of the intestine that will be best is the one which attaches itself as closely as possible to the functional examination of the stomach which can ultimately be combined with it; for it is indeed well known that not a few intestinal disturbances have their starting point in the stomach, and that it is under all circumstances necessary to have a definite knowledge of the work of the stomach before proceeding to the investigation of the function of the intestine. The examination of the functioning of the intestine must in the first place unfold a complete picture of the digestive activity; furthermore, it must inform us what groups of food-stuffs, in the given case, are especially defectively digested. Naturally in connection with the examination for undigested food-remains also the pathologic products of the intestinal wall, so far as they are recognizable in the stool (mucus, pus, blood, etc.), and the intestinal parasites must receive attention. Furthermore we must have in view as a goal to be sought, though it is still far distant, a separate examination of the intestinal activity according to its chief functions, motility, secretion, and absorption. Finally, we must seek to separate the disturbances of motility according to the chief section of the intestine (the large intestine and the small intestine), and the disturbances of the various secretions, that coöperate in intestinal digestion, stomach, liver, pancreas, and intestine.

The following presentation will show that thus far we have not yet advanced beyond the beginning of these problems of ours; they will, however, inspire us with the hope that we can make further progress on the way we have entered. After the discussion of the test-diet and the methods of examining feces, we will turn to the practical application of the functional examination in diseases of the digestive organs—the diffuse, inflammatory, and functional diseases of the intestine and its adnexa.

II. THE TEST-DIET.

Our Test-Diet.—Possibilities of Another Test-Diet.—Objections to our Test-Diet.—Irritative Effect of Milk on the Intestine.—Its Avoidance.—Advantages of our Test-Diet.

THE requirements which must be imposed on a suitable test-diet are very manifold; it must be so selected that it can be taken by healthy persons as well as by most of those suffering with intestinal disorders; it must be as free as possible, but not absolutely free, from waste matter, in order that the stimulus ordinarily furnished by the injesta should not be completely absent; it must be capable of supplying the minimum, at least, of calories required by the body (at rest) and must contain the three chief groups of food-stuffs in proportionate relation to each other, finally, it must be of simple composition, easy to make and uniformly prepared. Only when all these general requirements are fulfilled, the narrower selection with regard to the methods of subsequent fecal examination can proceed.

The first test-meal prescriptions, which *Strasburger* and I advanced in elaborating our fermentation test (5, 12), have suffered many variations, chiefly with a view of greater simplification (three forms of diet have become one), but partly through combination with the so-called "meat-test." Its principle—I mean the amount and kind of starchy food-stuffs served, which influence the fermentability of the excrement—has furthermore been retained. As it is superfluous to go through all the modifications of the test-diet, I state at once the prescription for the test-diet, which I use at present:—

In the morning: 0.5 liter milk [or, if milk does not agree, 0.5 liter cocoa (prepared from 20 Gm. cocoa powder, 10 Gm. sugar, 400 Gm. water, and 100 Gm. milk)],
to this 50 Gm. zwieback.

In the forenoon: 0.5 liter oatmeal gruel [made from 40 Gm. oatmeal, 10 Gm. butter, 200 Gm. milk, 300 Gm. water, 1 egg (strained)].

At noon: 125 Gm. chopped beef (raw weight) broiled rare with 20 Gm. of butter, so that the interior will still remain raw,
to this 250 Gm. potato broth (made of 190 Gm. mashed potatoes, 100 Gm. milk, and 10 Gm. butter).

In the afternoon: as in the morning.

In the evening: as in the forenoon.

This diet thus consists of: 1.5 liters milk, 100 Gm. zwieback, 2 eggs, 50 Gm. butter, 125 Gm. beef, 190 Gm. potatoes, and gruel of 80 Gm. oatmeal. It contains about: 102 Gm. albumin, 111 Gm. fat, 191 Gm. carbohydrates, or a total of 2234 calories (raw calories).[1] The test is generally given for three days, sometimes longer, at any rate until a stool is obtained, which comes with certainty from this diet. With a normal evacuation this occurs with the second defecation after the beginning of the diet. The patient as a rule does not need to take a special powder in order to mark off the normal excrement from the excrement of the former diet, for the normal excrement can generally be recognized at once by its uniform consistency and its lighter color. Where this is desired, or where the total excrement of three days is to be collected, I administer at the beginning and end of the test-diet a wafer with 0.3 Gm. powdered carmine, which passes the digestive canal unchanged and gives a distinguishing color to the corresponding part of the feces; only some very sensitive intestines are sometimes irritated by it.[1]

---

[1] The figures here given are computed after the tables of *Koenig*. Compared with the test-diet underlying our work on fermentative dyspepsia (19) the present diet shows 1 $^1/_2$ eggs and 20 Gm. sugar less; on the other hand 30 Gm. butter more (unimportant difference). Its advantages over the former consist in greater simplicity of preparation and in the greater proportion of milk used in the cooking of the diet. Only in the morning and in the afternoon is milk given alone and where this is not tolerated, it can be replaced by cocoa.

It is obvious that this test-diet does not represent the "only possible" or "only conceivable" form of diet for the functional examination of the intestine. To make such a claim never entered my mind. Within the above-described limits of the necessary conditions there are very many suitable diet lists for the clinical examination of the function of the intestine, and it may therefore again be expressly stated that we are here dealing with an altogether arbitrarily chosen standard. But what gives significance to just *this test-diet* is the fact that by means of it (or modifications of it) all fecal investigations were carried out by *Strasburger* and myself; that it is the foundation of all the tests to be described later; that it has furnished the basis of numerous quantitative analyses and experiments in digestion, which are given in our "Pathology of the Feces" (13) and to a certain extent form the basis of the entire coprology. *It would, therefore, be foolish to depart from this test-diet, or to desire to substitute another for it, without a cogent reason. Do such reasons present themselves?*

---

[1] **If diarrhœas exist and one wishes to collect the evacuation for a definite period, he must base the experiment on a greater number of days, for the sharp demarcation of the red from the uncolored parts is not possible, consequently an error creeps in, which can be overcome only by the extension of the test over a greater period of time.**

Thus far two objections have been raised against the test-diet. The one asserts that it is too difficult to be carried out, because its preparation in the hospital or in private practice occasions too much difficulty, and because most patients are not willing to content themselves for three days with such an insipid diet. The other criticises it as containing too much milk. Milk, it is said, does not agree with very many adults, it easily causes diarrhœa and is therefore unsuited for a "normal diet." Let us see how far these objections are justified!

The passive opposition of the kitchen force to all changes in the customary bill of fare is an existing fact with which every one must reckon who interferes with the food arrangements. Such an interference is, however, often urgently desired. Indeed, in the present condition of dietetics, I take it to be inevitable that there should occur a revision of the various (often quite obsolete) diet lists and a calculation of the individual diets and foods with regard to their content of calories and nutritive matter, so that the physicians should be in a position always to estimate the amount and the nutritive value of the food taken by the various patients. In such a revision the introduction of the test-diet among the diet prescriptions will, to judge from my experience, meet with no difficulty anywhere, for in itself the preparation is extremely simple, and the exact prescription of the ingredients makes it only easier for the kitchen force. And in private practice, the interest of patients in their treatment is for the most part so great that the more exactly the food is prescribed, the more carefully the prescriptions are carried out. I have really never encountered opposition to the test-diet from the patients themselves, except from such as had no gastric or intestinal troubles, whom we repeatedly used for purposes of comparison. Most of the patients in question were abundantly satiated, for some the quantity was even too large. This happens more often than the reverse. Taken strictly, it would be necessary in every perfect functional examination to adapt the quantity of food to the amount of calories needed by the various persons under examination, *i.e.*, first of all to their bodily weight; but this would, however, complicate the diet extremely. I am accustomed to adjust the individual variations of appetite first through the reduction or increase of the free amount of milk (in the morning and afternoon), because it is of little consequence for the subsequent fecal examinations; sometimes I permit the patient to leave some of the beef of his dinner, and, in the most unfavorable cases, some of the potato broth. Even through these changes the results are scarcely ever impaired. In general a certain latitude may be left in the test-diet in nearly all cases, *i.e.*, where it is only a question of qualitative investigations. Above all the amount of the prescribed starchy food-stuffs (100 Gm. zwieback, 80 Gm. oatmeal gruel and 190 Gm. potatoes) and the preparation (rather than the quantity) of the beef are important and must be closely adhered to. The meat must be at least partly raw (in the interior of the piece) for the result of the meat-test is based on this point. ***Zweig*** (14) and ***Strauss*** (15) have isolated my meat prescription from the test-diet and they give as a special "meat-test" 100 Gm. of raw chopped meat in connection with any diet at all. I consider the principle in such a mutilation of the test-diet false. I prefer to have the meat broiled rare over a strong fire, for the reason that to many persons raw meat is repulsive. Where the test-diet is to be taken for a longer period after the first fecal examinations are over, I permit the meat to be broiled more or less thoroughly according to the taste of the patients.

The *second objection* which concerns the milk, has a certain justification. It cannot be denied that milk is not tolerated by a number of persons suffering from gastric and intestinal disorders and even by some healthy persons, *i.e.*, such persons have diarrhœa at once, or—and this is to be especially emphasized—constipation. Very remarkable in this connection is the fact that it is almost always pure milk (raw or boiled) that presents these characteristics, whereas the addition of milk to other foods (milk cooked with anything) does not produce any such results. Indeed, many times merely a slight addition to the milk (cognac, cocoa, rice, barley water, starch) or even a different form of serving it (taking it in sups instead of drinking it, warming, boiling, etc.) suffices to prevent those disturbances. In most cases, moreover, the diarrhœa, or constipation represents only a temporary phenomenon, which stops of itself after 2 to 3 days. It is easily imaginable that in these cases, where the patient has not been accustomed to drink milk for a long time, the digestive canal and its flora require a certain amount of time to adapt themselves to the new diet. For the remaining cases we have as yet no satisfactory explanation of the peculiar effect of milk on peristalsis. The cause cannot lie in the ingredients of the milk itself, the mildest and least irritating food-stuff, which is tolerated by even the delicate digestive canal of the new-born babe. Perhaps in such persons it is decomposed more readily than normally and irritates the intestine through the increased products of decomposition, or the intestine, which is in itself already irritated is more sensitive than the healthy intestine to the normally formed products of decomposition.

The question is, in the first place, whether the incidental effects of milk on the intestine are indeed so numerous, that we need not include it in the test-diet at all. My own experience on this point is to the effect that the doubts expressed are very much exaggerated, and that under no circumstances are we to follow ***Rosenfeld*** (16) and summarily to put milk down as a substance irritative to the intestine. Among our numerous experiments with the test-diet extending over many hundreds of healthy and sick (partly seriously sick) persons, we have had to give up the investigation on account of the irritative effect of milk only very exceptionally (perhaps in 5 to 8 per cent.). I disregard here the cases, which are not very rare, where the diarrhœal or constipated effect extended only over the first 1 to 2 days, and I can only advise, that in case such disorders appear, the experiment should not at once be dropped, but prolonged at least for 4 to 5 days, before it is finally abandoned. The assertions of patients, that they cannot tolerate milk, etc., should not be given immediate credence; very often just such patients later tolerated milk very well. Where irritative effects really persist for a considerable time, so far as I can judge, it is very often a case of tuberculosis of the intestine, a disorder which is especially characterized by a capricious alteration of constipation and diarrhœa and by the fact that it is capable of being influenced by dietetic prescriptions only to a remarkably slight extent.

Nevertheless I admit that the desire to remove from the test-diet the possible injurious effects incidental to milk has its justification, and in the present form of the test-diet, I therefore have one-third of the entire amount of milk cooked with the food (I have never seen any irritations resulting from the milk contained in the oatmeal gruel or potato broth). Instead of the other two-thirds, in cases where disorders arise or the sick are unwilling to take milk, I permit the patients to take a corresponding amount of cocoa boiled with only a little milk. The evacuation is thereby colored a somewhat brownish red, and on microscopic examination it shows numerous residue of cocoa nibs, along with the scanty spelt remains coming from the oatmeal gruel and the zwieback. Only quantitative (digestion) experiments must naturally be abandoned. Instead of this change, the proposal, made by me (17) for therapeutic purposes, to reduce the decomposability of the milk through the addition of very slight amounts of pure salicylic acid, can be used for the test-diet. For this purpose 0.3 Gm. of pure salicylic acid is boiled with the daily portion of milk ($1\,^1/_2$ liters) in the morning before the preparation of the diet, in the following manner: The salicylic acid powder is first stirred up thoroughly in some cold milk, then this sample is added to the daily portion, stirred well and boiled once. The milk does not thereby lose its character or taste, neither does it coagulate, if thus dealt with. As a result it takes on a certain resistance to the influence of bacteria, which can be proved directly, as *Strasburger* (18) has shown, by the diminution of fecal bacteria.

I believe that these two ways will practically always suffice to remove the disagreeable features, which—as has been said, in rare cases—might grow out of the milk contained in the test-diet. Where this is not the case, I should prefer to give up the functional test altogether, rather than to omit the milk from the test-diet. For this I have good reasons, as I do not believe that a suitable test-diet can be combined at all, leaving out milk. For the amount of starch and of meat in my test-diet does not allow an increase for the purposes of fecal investigations, and under these circumstances how are we to produce the needed amount of calories by means of sugar, eggs, or other skillful nutritive preparations, without striking upon new difficulties? Turn the matter as we may, a uniform test-diet that would be suitable for most cases in question, seems to me impossible without some milk as an ingredient. Besides, and this is a second important reason, milk is and remains the food-stuff which, possessing a satisfying nutritive value, claims relatively the least chemic and mechanical digestive work. It is the food-stuff to which the disturbed intestine must therefore accustom itself again, if this is at all possible, in order to return to a healthy state. For me it is the first therapeutic principle in all catarrhal and functional disturbances of intestinal activity to strive to make milk or milk preparations (buttermilk, kephyr) tolerated again, and I regard the test-diet therefore not only as a diagnostic, but also at the same time as a therapeutic aid, which in many slighter affections alone suffices to bring about a cure. *Strasburger* and I (19) have already pointed this out, when we described fermentative dyspepsia, and my further experience is to the same effect.

Summing up all this, I come to the conclusion that even the large milk content of the test-diet is no cause for changing it. As it is, it has stood the test, and as long as we can find no better one to put in its place we will quietly adhere to it.

III. THE EXAMINATION OF THE FECES.

As soon after the beginning of the test-diet as the evacuation has taken on the easily recognizable characteristics of the "normal excrement" (light brown color, uniform consistency), which generally occurs in the second or third defecation, we proceed with the investigation. I do not regard it as superfluous to remark here that when patients cannot deposit their excrement in suitable transportable vessels (commodes, etc.), there should be placed at their disposal for the purpose glass jars about 15 centimeters high and 8 centimeters in diameter, with screw-top glass covers. These filled glass jars should be sent for examination immediately after evacuation. This investigation should be carried out on feces as fresh as possible. If carried out in the manner subsequently to be described, it requires at most ten minutes and only very simple apparatus, so that in all respects it can be placed alongside the usual examination of the urine and of the stomach-content.

A. Macroscopic Examination.

After determining the consistency, color, and odor, the *macroscopic investigation, the most important part of the whole procedure,* which often alone suffices to enable the experienced observer to form a judgment, is begun. For this purpose the excrement is first stirred up thoroughly with a wooden spatula, and a small amount of about the size of a walnut is transferred to a glass mortar (of about 12 centimeters in diameter). In this it is ground up as fine as possible with the addition of at first a few cubic centimeters and gradually more distilled water (until it is of fluid consistency). The grinding up must be done carefully, so that no cohering excrement particles remain. The ground up specimen of excrement is then spread over a flat black plate or a large glass vessel in as thin a layer as possible and account is taken of all the elements that are still recognizable in it with the naked eye.

This method of macroscopic investigation (by grinding up the excrement) is the only correct and suitable one; it is to be preferred by far to stirring it up or washing it through sieves (*Boas* and others) (20). The latter has a value only when parasites or gall-stones are being exclusively sought for. *In normal digestion there ought to appear very few brown points (smaller than pinheads) (chaffy remains of the oatmeal gruel, perhaps remains of cocoa nibs), the nature of which is ultimately explained by microscopic investigation.*[1]

Under pathologic conditions there appear:—

1. *Mucus, in larger and smaller, even very small flakes.* In the process of grinding up the excrement, unless special force is used, the mucus is scarcely broken up, and appears in its original form. If it is in larger shreds, or strips, tubes, moniliform, etc., as in the case of enteritis membranacea and colica mucosa, the recognition of it will scarcely ever occasion difficulties. Nevertheless, we should not forget that, if the mucus of the large intestine is thickly set with worn-out cells and fat-bodies, it may lose its original glassy character and soft consistency altogether and assume a white or brown color, with a gummy or even a

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[1] **When the patient has eaten fruit or other vegetables before the test-diet, there occur in the excrement for many days thereafter solitary remains of the same as little points which grate on rubbing. Larger crystals of ammonio-magnesium phosphate (see under 6) can also give the sensation of grating when rubbed.**

leathery hardness. The smaller the flakes of mucus, the more difficult is it to recognize them, although, according to my experience, there is *no form of mucus that is found mingled with the feces, which on thorough inspection cannot be recognized even with the naked eye—especially if some of the ground-up mass is placed on a glass plate and held up against the light* and seen as glassy, transparent flakes, occasionally colored yellow by bilirubin, with irregular, ragged outlines. The mucous particles intermixed with the feces, earlier described by *Nothnagel* (4) and recognizable only by microscopic examination, the so-called "hyaline mucus-islands" and the yellow "mucus-granules" do not consist of mucus, as I have proved (13, 21). If any one is in doubt whether any little flake is really mucus, or wishes to make sure of the more minute structure of the mucous flakes (amount, kind of cells, parasite eggs, amœbæ, crystals of bilirubin and Charcot-Leyden crystals, bacteria, etc.) which is often desirable, he can place a little flake under the microscope and examine it—best without staining—with low and later with the *highest* powers.

2. ***Flakes of pus, blood, tape-worm proglottides and other parasites, stones, remains of egg shells, and other foreign bodies.*** **These will not be discussed further here. For their demonstration and semiotic significance. Cf. "Die Fäzes des Menschen," I., p. 37ff.**

3. *Remains of connective tissue and tendons from the chopped meat eaten.* These are frequently found and can at once be distinguished from the mucus by their whitish yellow color, their thread-like appearance, and their solid consistency. In doubtful cases a thread is placed under the microscope and treated with acetic acid, in connective tissue the thread-like structure disappears, whereas in mucus it then first appears. *Completely isolated little tendon fibers are occasionally found even with normal digestion. As*

soon as they appear more numerous and larger, a disturbance of digestion, namely of gastric digestion, is involved.

4. *Remains of muscular tissue.* Chiefly very small, brown colored rods, looking like splinters of wood; can be broken up by pressure and show muscular structures under the microscope. *The muscle remains point to a disturbance of intestinal digestion.* Occasionally remains of connective tissue and of muscle occur combined as meat remains.

5. *Potato remains.* The potato remains appear like sago grains, glassy, transparent granules, and can easily be confused with flakes of mucus.[1] For examination, their globular appearance and larger consistency causes them to project above the level of the thinly spread layer of excrement, whereas the mucus spreads out flat. Crushed, under the microscope, they show the potato cells with remains of starch granules that can be colored blue with iodine.

6. *Large crystals of ammonio-magnesium phosphate.* These are present in rare cases, in putrefying, mal-odorous stools; they grate when the excrement is ground up in the glass mortar, and can easily be recognizable by their well-known coffin-lid shape and chemical reaction (solution in all acids).

B. Microscopic Examination.

The microscopic examination serves for the most part to complete the macroscopic; by means of it alone pathologic conditions are seldom disclosed. At all events it is well to carry it out in the following way: If the parts that are recognizable with the naked eye are eventually to be examined with the aid of the microscope, three preparations are made of the feces that was originally well stirred up—if it is too hard, a little water is added—*i.e.,* small particles are placed respectively in the middle and on either end of a slide. Without adding anything to it, the *first* is crushed into a very fine layer with the cover-glass. The *second* is stirred with a small drop, 30 per cent. (official), acetic acid, and heated for a moment over the flame (until it begins to boil), and then covered with the cover-glass. The *third* is rubbed up with a small drop of a strong solution of iodo-potassium iodide (iodine, 1.0; potassium iodide, 2.0; aq. dest., 50.0).

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[1] Former and even late investigators erroneously described these as mucus (13, 22).

The normal excrement examined in this manner gives the following result: In the first preparation without any addition (cf. Fig. I) there is seen a detritis consisting of the smallest granules, globules, bacteria, etc., of the most varied kind, the more minute details of which cannot be recognized in ordinary inspection with strong dry lenses (Fig. I*f*). The larger bodies imbedded in these are:—

Isolated fragments of muscle fibers, yellow in color, with rounded edges, flake-like structures, with indications of cross striations now and then (Fig. I*a*); further

scattered smaller and larger yellow salts of calcium, flakes consisting of fatty acid salts of calcium with irregular borders, bright and deep yellow in color (Fig. I*b*), and

colorless soaps (Fig. I*c*);

isolated potato cells, either empty or with agglomerated remains of a content, which can no longer be differentiated (Fig. I*e*);

scanty chaffy remains from the oatmeal gruel, often recognizable with the naked eye as little dark flakes (Fig. I*d*);

finally perhaps (where cocoa is taken instead of milk) remains of cocoa (Fig. I*g*).

In the second preparation which serves to melt all the fat-remains temporarily and thus to furnish a view of the entire fat-content, there is seen, after cooling, *small flakes of fatty acid* scattered over the entire preparation. The larger salts of calcium and soap flakes have disappeared. If the preparation is again carefully heated over the flame and placed under the microscope while still hot, the small flakes of fatty acid are melted into drops, which congeal very suddenly, as the preparation cools again. In the third preparation, which appears brown in color, the potato-remains ultimately assume a *violet* shade (not a blue) and *isolated* fungus-spores, blue-violet in color (Clostridium butyricum?) appear.

The following conditions are pathologic (cf. Fig. II):—

1. *In the native preparations:*

Fragments of muscle in larger number and better state of preservation (Fig. II*a*).

These appear as longer bundles with cross striations and sharp edges, or as a collection of irregularly outlined homogeneous flakes (yellow granules).[1]

Drops of neutral fat (Fig. II*c*).

Needles of fatty acid and soap. Numerous little crystals constituting the largest part of the mass (Fig. II*b*).

Numerous groups of potato cells with more or less well-preserved starch cells.

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[1] The *digestion test* of the feces previously described by me, which depended on a measurement of the quantity of undigested (macroscopic and microscopic) muscle remains, I abandoned later as too detailed and superfluous. The appearance macroscopically of muscle remains suffices decisively to prove a disturbance of intestinal digestion. The microscopic finding of many fragments of muscle fibers serves to supplement the same, only in rare cases can the diagnosis be based on it alone.

Parasite eggs (it is self-evident that mucus, connective tissue, pus, and other macroscopic parts appear).

2. *In the acetic-acid-preparation:*

massive fatty acid flakes, so that in comparison the remaining components form a very slight part.

3. *In the iodine preparation:*

blue colored remains of starch granules (in potato cells or free) (Fig. II*d*);

blue or violet fungus spores and thread-like bacilli (Fig. II);

yeast cells (which are colored yellow by iodine) (Fig. II*f*).

C. The Chemic Examination.

Of chemical tests only the following need to be considered for practice:—

The testing of the reaction, the sublimate test, the fermentation test, and perhaps the examination for dissolved albumin.

For the reaction test a strip of red and blue litmus paper is placed on (not in) the excrement, which had been ground up with distilled water (for the macroscopic examination). Normally the reaction is amphoteric or *at most feebly* acid or *feebly* alkaline.

The *sublimate test* (13, 23, 24) consists in placing some of the same feces, that had been ground up with water, in a glass vessel filled with a concentrated watery solution of $Hg\ Cl_2$, stirring it and leaving it stand (until the next day). The normal feces is colored red by this (hydrobilirubin), and the more intensively, the fresher and less decomposed the excrement is. Particles colored green, even if microscopically small, are pathologic. They show the presence of unchanged bile pigment (bilirubin).

The *fermentation test (incubator test)* as I perform it now, has been very essentially simplified as compared with the original fermentation test. From the fresh excrement (which has been stirred up, but not yet thinned with water) a small amount[1] of about the size of a little walnut is taken with one of the wooden spatulas and put into the lower vessel of *Strasburger's* fermentation tube (Fig. III*a*).[2] (In hard stools less is taken; in soft more; in fluid stools the lower vessel is filled entirely. In this it is stirred up with water and the rubber stopper is put on, care being taken to exclude air bubbles. The rubber stopper is taken from the little tube *b* and the tube filled with tap-water. This is then closed by putting on the stopper with the vessels connected with it, *a* (filled) and *c* (empty); *c* has an opening on top and acts as an ascension-pipe *(Steigrohr)*. Note is taken of the figure indicating how far the connecting glass tubes enter into the lumen of the three vessels. When the apparatus is put together, it is placed for twenty-four hours in an incubator heated to 37° C. If gas is developed from the feces within this time, this collects in *a* or *b* and a corresponding amount of water is driven into the ascension-pipe *c*. The height of the water in the ascension-pipe is noted, the vessel *a* is opened, and the reaction of it is tested with litmus paper, and compared with the reaction before the test was instituted.

Normally only very little gas is formed in the incubator test and the original reaction of the feces undergoes no important change. If gas is developed to such an amount that one-third or more of the ascension-pipe is filled with

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[1] It should be 5 Gm., however, the estimated amount is sufficient for our purposes.

[2] Can be obtained through *C. Gerhardt*, Utensilienlager, Bonn, Bornheimerstr.

water, then pathologic conditions are involved, and if at the same time the reaction has become distinctly more acid, carbohydrate fermentation (the positive result of the fermentation test), has occurred; if it has become distinctly more alkaline, albumin putrefaction has occurred.

In the former case the opened glass *c* gives off the odor of butyric acid, in the latter case it gives off a very decided putrefactive odor. The color of fermenting feces is generally brighter, that of putrefying feces is generally darker. The suspicion of pathologic fecal fermentation can often be confirmed by the mere inspection of the original feces, namely, if they appear light brown and foamy, react acid, and smell of butyric acid.

In order to examine for dissolved albumin[1] the remainder of the specimen of the excrement which has been ground up with distilled water for macroscopic examination is passed through a double filter. The filtrate is clarified by shaking it up with diatomaceous earth and filtering it again, and then strongly acidified with acetic acid, until the precipitate which at first forms (nucleoproteid) is again dissolved, and further mixed with a drop of potassium ferrocyanid solution. If it thickens *definitely*, it indicates albumin. From the positive result of this test, we cannot, for the present, draw any further conclusion than, that there exists a diseased disturbance of digestion. Of what kind, must as yet remain undetermined.

D. The Bacteriologic Examination of the Feces

will not be discussed here, as we are dealing only with the lighter affections of the intestine, and the differentiation of special forms of ailments on the basis of methods of bacteriologic investigation has not yet proved successful. We merely mention that the proof of tubercle bacillus in the feces permits the diagnosis of intestinal tuberculosis only when it is

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[1] The method here described is based on experiments which Dr. *O. Simon* carried out in my laboratory not long ago. The procedure offered by *Ury* (74) is too detailed, and also not entirely free from objection. Dr. *Simon* will shortly publish his results elsewhere.

(Dr. *Simon* has since published his results in the Archiv f. Verdauungskrankheiten, page 197, 1904.)

obtained in isolated flakes of mucus, washed off with water. For tubercle bacilli contained in the fecal mass may originate in swallowed sputum.

At the conclusion of this section I might again point out that the fecal investigations here described—presupposing some practice—*as a rule,* do not require more than ten minutes. On the next day nothing remains but the brief inspection of the fermentation and sublimate tests. For the most part I make all the tests in order, with perhaps the exception of the test for dissolved albumin. In the repetition of the examination we can restrict ourselves to those tests which have furnished pathologic results. It is important to repeat the tests in all cases, which are not entirely clear, for it is a question that may alter from day to day, just as it is the case in the investigation of the stomach-contents. *Just in this fact, that the fecal investigation by means of the test-diet, shows the ephemeral variations of digestion which are lost in experiments of digestion, I see one of the chief superiorities of this method.* All well know how quickly diarrhœa and constipation, good and bad digestion, can follow each other. I do not understand, therefore, how it is that various investigators, who have examined our fermentation test, have concluded from the fact that the results vary that our method is impracticable. If we wish to find objective symptoms for the lighter disturbances of digestion, which through our former inadequate methods of investigation exhausted themselves in subjective feelings, the test-diet has to be administered often for a longer period of time and the fecal investigations made daily. Then often through temporary pathologic conditions (fermentation, mucus, connective-tissue-remains, etc.) we will be shown that something is here out of order, and wherein that something consists.

The longer I work with the test-diet and these simple methods of investigation, the more indispensable have they become for me, and I do not doubt but that would be the experience of every one who would adopt them with an unprejudiced mind.

IV. THE SEMEIOTIC SIGNIFICANCE OF PATHOLOGIC FINDINGS IN THE FECES.

Although this treatise is concerned principally with the functional examination of the intestine, *i.e.,* with the question why certain food remains in a given instance pass through undigested, we must include in our investigations other pathologic findings in the feces, above all mucus, unchanged bile pigment, and the phenomena of fermentation or putrefaction.

1. Mucus.

The appearance of mucus in the feces indicates the existence of an inflammatory condition of the mucous membrane, it is the sole reliable sign of this. To this rule there are only two exceptions, and even these do not always hold: a thin mucous coating, that on drying looks like varnish, is occasionally found on the surface of scybala in difficult defecation, without a true catarrh of the rectal mucous membrane, and in the so-called mucous colic, in which spasmodically large masses of glassy mucus are discharged, the nervous overproduction of mucus exceeds the local changes to such an extent that the aspect of the disorder removes it from the bounds of ordinary catarrh [*Nothnagel* (22)].

For all remaining cases the following is of importance:—

(a) With the exception of few instances the mucus that appears in the feces originates in the large intestine, and it is supposed the higher the parts attacked by the inflammation, the smaller are the individual flakes and the more uniformly distributed are they found in the excrement.

(b) The assumption that it originates in the small intestine is admissible only in case of very liquid feces and only for the smallest mucous flakes, if these on microscopic examination are thickly inter-

spersed with bacteria and half digested cells, or cell nuclei in characteristic arrangement. Bilirubin coloring of the mucous flakes (sublimate test), or the presence of fine bilirubin crystals under high power (typhoid fever, tuberculosis, cholera, severe acute catarrhs) corroborates the above.

(c) The more thickly the mucus (in the microscopic preparation) is interspersed with cells (especially round cells)—whitish coloring can also arise if fat is enclosed—the more intense is the inflamed condition of the mucous membrane. In ulcerative processes there is found almost always only purulent mucus in the feces, scarcely ever pure pus.

(d) Ulcers and catarrhs, especially if they are high up, might exist without an evacuation of simple or purulent mucus or blood. The products of the inflammation are then dissolved by the digestive fluids just as the food ingredients.

2. Unchanged Bile Pigment (Bilirubin) and Variations in the Hydrobilirubin Content.

Normally after the passage of the intestinal content through the ileo-cecal valve the bile pigment is, through the processes of reduction, connected with the putrefaction that here sets in, converted into hydrobilirubin. The hydrobilirubin assumes a red color in the sublimate test, unchanged bilirubin becomes green.

(a) A green coloring of the entire excrement or of separate parts of it (chiefly of the muscle fragments and of the mucus) is—aside from the feces of newly-born babes or sucklings that scarcely need to be considered here—pathologic. It indicates a too rapid passage of the intestinal content or a defect of the normal processes of reduction for other as yet unknown reasons. Usually when the entire stool is colored green by $HgCl_2$ the small intestine is also affected. The diagnosis that the small intestine is affected becomes certain only in connection with other signs, namely, with characteristic mucous flakes (see I*b*) [*Schorlemmer* (26)].

(b) The sooner after evacuation the feces are treated with $HgCl_2$ the more intense is the red coloring. If the same phenomenon appears incompletely (a dirty red coloring) when fresh, then in most cases abnormal processes of decomposition are involved.

(c) Complete absence of the red (and green) coloring in the sublimate test occurs in fatty stools, when the bile is completely shut off from the intestines (the $HgCl_2$-test proves the complete obstruction of the biliary duct), and in rare instances in the so-called clay-colored stools without icterus (temporary suppression of the bile?).

3. Insufficient Digestion of Fat.

To answer the question, whether in a given instance the fat has been sufficiently digested or not, we have no uniform standard even when we use the test-diet. Inasmuch as every stool, even the fasting stool, contains fat—the normal excrement, about 23 per cent. of the dry substance—and inasmuch as too great pathologic significance is not to be attributed to small variations of fat-digestion, it is only a *considerable* increase in the fat in the feces that need be considered practically.

This is comparatively easy to recognize in macroscopic investigations, by the clayey appearance and the formation of a membrane on the surface, while the feces is being rubbed up with water, and in the macroscopic investigation by the large increase of flakes of fatty acids in the heated acetic acid preparation (Preparation 2), and eventually by the presence of drops of neutral fat or of fatty acid and soap needles in the native preparation. Fatty stools are of a light or whitish color, are abundant and of acid reaction. I do not believe that a real fatty stool is overlooked easily, and therefore do not regard a special test for its recognition necessary.

Fatty stools may occur:—

(*a*) In deficient secretion or outflow of bile. In this case there appears in the sublimate test little or no red coloring of the feces. Usually icterus exists at the same time.

(*b*) In the disturbances of the pancreatic secretion. In such cases the fat frequently appears in the form of drops (neutral fat), and at the same time muscle-remains, recognizable macroscopically, are found in the feces. Hydrobilirubin represents, unless the flow of bile is checked, a somewhat frequent combination.

(*c*) In disturbances of the intestinal digestion. This is comparatively rare, at any rate it is not a correct rule that in intestinal disorders the fat absorption suffers the most. In extreme intestinal tuberculosis, intestinal amyloid, tabes mesaraica, and similar conditions it is true the fat absorption suffers more than the albumin digestion [*Weintraud* (27)], but a constant relation does not obtain here. Purely functional disturbances of fat digestion occur [*Salomon* (28)]. The recognition of all these conditions is possible only through the exclusion of *a* and *b*, while at the same time attending to other pathologic signs (mucus, processes of decomposition, etc.).

Eventually the fact that the improvement sets in after pancreatic preparations have been administered for an intestinal affection, may be of value. This will be discussed later.

4. Meat-Remains.

Through extended experiments I have duly determined (29), that if 100 Gm. of beef, cut up fine enough, is taken, under normal conditions, no macroscopically recognizable meat-remains reappear; whereas, this is often the case in disturbances of digestion. On this is based the "meat test" interwoven into the test-diet, which, as already mentioned, has later been taken out of the test-diet by *Zweig* and *Strauss* and recommended as a special test. If care is taken to have the beef chopped fine enough, and if, in part, it is left raw, these rules of diagnosis follow:—

(a) *If connective tissue-remains appear in the feces it is a sign of disturbance in the stomach digestion.* Of all the digestive secretions, as I have shown, the stomach juice alone can digest *raw* connective tissue. The stomach alone is at fault if connective tissue appears in the feces. In most cases it is a question of hypochylia or achylia (as an independent disorder, in catarrhs, the development of cancer, etc.), and the disturbance may become so severe that the connective tissue is quantitatively discharged as a fine down intermingled with the whole stool. In many of the above conditions an increased motility plays a part. In hyperacidity, occasionally remains of connective tissue are found, and then, if the motility is normal or retarded, we must go back for explanation to an insufficient secretion of pepsin, which, according to recent investigation, may well be combined with increased HCl-secretion (31). At any rate the disturbances of the activity of the stomach, which are indicated by remains of connective tissue, are not uniform. On the other hand the reaction of connective tissue is extremely valuable in many instances and has often led me to the right path. To this I owe the discovery that many more intestinal disorders than are generally supposed have their starting point in the stomach. This will be taken up later.

(b) *If macroscopic muscle remains appear in the feces, this is a sign of a disturbance of intestinal digestion.* The stomach shares in the dissolution of muscle only to a slight percentage. By far the largest part falls to the share of the intestine (17, 32). If this does not perform its functions normally, whether it be that the pancreatic juices are not sufficiently secreted or that the highly increased peristalsis does not allow time for digestion, or that a primary disturbance of absorption leads to decomposition and thereby secondarily to diarrhœa, the muscle fragments may reappear. *It follows from the above that the disturbance* *of a definite function of the intestine cannot be inferred from the appearance of muscle fragments alone; we might, however, say that the disturbance is located in the small intestine,* for the large intestine is only a reservoir for after-digestion, and in it the solution of pieces of meat no longer occurs (33).

If the particular disturbance is not so severe as to cause the macroscopic muscle-remains to appear, under certain circumstances, an increase of microscopic muscle fibers similarly denotes a digestive disorder. Conclusions which base themselves on microscopic investigation alone are therefore to be used with caution, nevertheless I have myself seen intestinal disturbances, which consisted *exclusively* in a large increase of microscopic muscle-remains.

(c) *Remains of connective tissue and muscle alongside each other or together as meat-remains, indicate, disturbances in which stomach and small intestine participate.* In an acute form this meat lientery sometimes appears in acute gastro-intestinal catarrhs. Through Mr. *Brink* (34) I have had such cases described, and I will later refer back to them.

5. Carbohydrate Remains (Fermentative or Incubator Test).

Of the carbohydrates contained in the test-diet, the milk sugar and the zwieback—leaving out of account the very rare severe disturbances—are absorbed without any residue. The potato broth serves essentially as a criterion of satisfactory starch digestion. The amount of it is so chosen that normally only a small number of empty potato cells (taking on a weak violet color, when stained with iodine) appear in the feces. If the starch digestion is interfered with, a phenomenon which occurs even as an isolated disturbance (intestinal fermentative dyspepsia), the following combination is generally found: a light brown, foamy appearance of the excrement, acid reaction, and numerous microscopic potato cells with a content that take on a blue color when treated with iodine, and perhaps granules containing spore and thread-like fungi, the positive result of the fermentation test. Lighter grades of disturbed starch digestion show themselves through only a part of these symptoms. *Defective starch digestion has its seat in the small intestine, it depends for the most part upon a disturbance of the secretion of the intestinal juice and* per se *signifies (without any other alteration of the condition of the feces) no severe disturbance* (19). Only the positive result of the fermentation test is of value for diagnostic purposes. The conclusion, that in the absence of the formation of gas, the digestion of starch is normal, is not a reliable one. For it may occur that the carbohydrate fermentation in the incubator test may exhaust itself in the formation of acid products (without at the same time forming gas), *e.g.*, in fatty stools; it may happen that the carbohydrate fermentation is overtaken by processes of putrefaction that take place simultaneously (35, 36). Under certain circumstances the microscopic finding alone furnishes sufficient proof (numerous potato cells with iodine reaction, granules-containing microbes).

6. Relation of Fermentation to Putrefaction.

(Course of the processes of decomposition in the incubator test.)

The appearance of acid fermentation (CH fermentation) is not the only alteration which the feces may undergo in the incubator test. Sometimes gas also forms with an increasing alkaline reaction; the color of the excrement then becomes darker, the smell intensely putrid (albumin fermentation). In general in carbohydrate fermentation gas is formed more rapidly and in larger quantities ("quick fermentation") ("Frühgärung"); in albumin fermentation it is formed later in smaller quantities ("slow fermentation") ("Spätgärung") (37). Slight degrees of one as well as of the other form occur under normal conditions, even when the "normal excrement" shows practically no changes in the incubator test; only higher degrees are pathologic. The limit of one-third of the height of water in the ascension-pipe *c* of the fermentation tube has been determined empirically; it is not to be taken absolutely. Pronounced changes of reaction to one or the other side can under certain circumstances be just as important for judging the processes of decomposition as the gas formation. Especially to be considered as the determining characteristics for putrefaction are, definite alkaline reaction and odor of putrefaction in the freshly evacuated feces, a darker coloring, and the appearance of coffin-lid crystals (see above).

The manifestation of definite albumin putrefaction in the incubator test allows us to infer severe disturbances of digestion, disturbances which are for the most part connected with anatomic changes in the intestinal mucous membrane. The cause seems to be that the pathologic secretions of the intestinal wall (mucus, serum, pus) when mixed with the feces are especially liable to decomposition.[1]

On the contrary, however, experience teaches that where albumin is imperfectly digested for purely *functional* reasons, even if dissolved albumin is present in the excrement, only rarely do higher degrees of fecal putrefaction appear.

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[1] **Perhaps because they are usually the carriers of special putrefactive excitants.**

The fermentation test forms only one part of the incubator test which—speaking generally—tells us as regards the direction of the processes of decomposition in the feces. This orientation is, however, present only, when the one or other process (CH fermentation or putrefaction) predominates decidedly. Where such is not the case, where a combination of both processes exists, we must for the present do without any indication.[1]

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[1] The exposition of the "fermentation test" here given and its diagnostic signification differs in many respects from our original conception, even from that last laid down in the "Fäzes des Menschen." I have assured myself more and more in the course of time that it possesses a more general value than merely to indicate a possible diminution of starch digestion. The assumption, that the "fermentation test" represents a universal test of the feces, which indicates even the mildest digestive disturbances, has led some scholars who occupied themselves after us, to entirely false conceptions (25). At no time have our statements given rise to such a comprehension.

Since in the "incubator test," as I again desire to emphasize, only the *decided* changes toward the one or other direction (acid fermentation, putrefaction) are turned to account diagnostically, and an exact limit between normal and pathologic can indeed scarcely be established at any time, I have given up the weighing of 5 Gm. excrement (medium consistency) for the fermentation test and rather placed the limit of gas formation somewhat higher (one-third height of the ascension-pipe in place of the earlier one-fourth). I therefore lay the greatest importance on the simultaneous change of reaction (acidity of the excrement). In doubtful cases one repeats the test with the next evacuation. For the experienced the changes in reaction alone can assume a meaning; nevertheless one should bear in mind that fatty stools also show acid reaction (on account of the rich content of free fatty acids). In *putrefaction* the height of gas formation is even less significant than the remaining signs.

V. RETROSPECT AND FURTHER PROBLEMS OF THE FUNCTIONAL EXAMINATION.

Incompleteness of the present functional examination.—Necessity of a better method for examining the pancreatic secretion and of a method for determining the length of time food remains in the small intestine.

It would be rash to assume that the methods which have been described in the preceding sections, the test-diet and the fecal examinations based upon it, are at present adequate for a satisfactory functional examination of the intestines and the digestive glands connected with them. I myself feel that we have not as yet advanced beyond the beginning, and that the goal before us, the obtaining of a functional examination analogous to that of the stomach, is still in the distance. What we have attained is a uniform basis of investigation and a tolerably sharp demarcation of certain digestive disturbances originating in the stomach from others having their origin in the intestines, and further, a separation of the latter according to the groups of foods (meat, fat, starch) which the disturbance in the special instance affects. We have not yet succeeded, although it is an absolute necessity, in assigning any given digestive disturbance to any one or more of the three chief functions of intestinal activity: secretion, motility, and absorption, and in localizing the disorder according to the different secretions (bile, pancreatic juice, intestinal juice) or parts of organs (small and large intestines) affected. At present only in a few cases can we go so far. The difficulty here lies in the extremely complicated interaction of the three functions named as well as of the different secretions and intestinal divisions. It is indeed evident that a disturbance of secretion must in each instance bring about a disturbance of absorption. The same is true for increased peristalsis. Disturbances of secretion as well as of absorption lead to an increased decomposition of the intestinal content which may have as a consequence an increased peristalsis, etc. In the stomach the conditions are very much simpler, because absorption hardly comes into consideration, and motility and secretion are independent of each other to a much higher degree. In the intestine we have, and this complicates the matter very much, for each group of food-stuffs several secretions (stomach and pancreas for albumin, gall and pancreas for fat, saliva, pancreas, and intestine for starches). Our present means of direct quantitative determination of the various digestive secretions are still very incomplete; we must, for the most part, infer them indirectly from the disturbances in absorption, which they cause. Finally we are altogether without a suitable standard for judging motility. What we can learn concerning this function from the existence of diarrhœa or constipation, and from the length of time food remains in the digestive tube extends almost entirely to the movements of the large intestine. We know nothing of the extent to which the small intestine participates in diarrhœa or constipation in a given instance, and yet it is just the motor activity of the small intestine which is for us of much greater interest than that of the large intestine. Even in such obvious disturbances as catarrhs or ulcers, the problem of determining whether they are situated in the large or small intestine often meets with insurmountable difficulties. How much more is this the case with purely functional disturbances?

These considerations suffice to show us where our activity must be directed in the future if we are to make progress. *In the first place, the problem is to find a simple method which will permit the direct determination of dis-*

turbances in the secretion of the most important of the digestive juices, the pancreatic juice. If we had such a method, the recognition of disturbances in the other secretions, the intestinal juice and the bile, would come by itself, for both of these have quite different duties to perform, and, moreover, in the bile pigment, we have even now an important aid in determining the biliary secretion. Of course, attempts have not been wanting to test the pancreatic secretion, but all, even the very ingeniously devised method of *Sahli* (38, 39) have given no satisfaction. As I shall discuss these conditions later, I shall omit a detailed criticism of *Sahli's* procedure here. I restrict myself to the mere mention that the glutoid capsule test in its present form can lay claim to diagnostic value only when it gives a negative result, *i.e.,* beginning of the iodine reaction at the right time in the saliva excludes any affection of the pancreas. The positive result would, however, be of greater value to us. I myself have taken the trouble to devise such a positive test from the absence of digestion of the nuclei (a function that belongs exclusively to the pancreas) in pieces of meat served especially for this purpose, and I believe I may assert that, although my investigation in this respect is not completed, it does in fact furnish the desideratum, but—and this large "But" confronts us in all our results with regard to the pancreatic secretion—a disturbance of the pancreatic secretion can be shown by it only when the gland is destroyed or degenerated to the last degree. But then our subtle methods can no longer be of much avail, for then the affection can be recognized by other more obvious signs. Slight disturbances of the pancreatic secretion, the occurrence of which, in accordance with the analogy of the stomach, we might expect to determine, we have hitherto been unable to prove in any way. But let me not anticipate my later discussions.

In the second place we need a simple way of measuring the length of time the food remains in the small intestine. If we had such a method, in connection with the easily determinable length of time the food remains in the entire digestive canal and with the determination of the motor power of the stomach, we could localize exactly all disturbances in motility and so would make a great step forward. It is remarkable that hitherto no efforts have been made to solve this problem. Even my own detailed efforts extending over many years have unfortunately been without result, but that might be no reason for abandoning them altogether.

I take the liberty of describing here very briefly the way in which I had hoped to penetrate into this obscurity. As has been pointed out above the valve of *Bauhin* (ileo-cecal) is the point where the putrefaction of ingesta begins (33). In the small intestine putrefaction of albumin arises only under special pathologic conditions. Immediately beneath the valve powerful processes of reduction set in with putrefaction. Through these bilirubin, *e.g.*, is converted into hydrobilirubin. It is such processes that produce out of the cholesterin of the food and the bile a substance, constantly present in the intestines—coprosterin—a substance differing from cholesterin only through the addition of 2H-atoms (40). Cholesterin has the characteristic of combining readily with iodine and bromine, whereas this characteristic is wanting in coprosterin. On this chemic fact I founded my hope. I assumed that if we succeed in finding an iodine or bromine combination of cholesterin, which would pass the stomach and small intestine unchanged, it would have to be split at the *Bauhinian* (ileo-cecal) valve, because the coprosterin which arises there from the cholesterin no longer combines with the halogens. The freed iodine or bromine would then have had to show itself in the saliva, exactly as in the glutoid test. Dr. *Nerking*, who at my instance took up the preparation of the cholesterin combinations needed, succeeded, after great exertions, in obtaining suitable combinations, but the event proved that these combinations passed the digestive canal unchanged, that they were too firm to be split by the process of reduction in the small intestine. And this disappointment is not the only one which I have met with. I will omit an account of the others, but I do not give up the hope that we will at some time succeed in solving this problem in connection with the test-diet. At any rate, it must first be solved before further plans can be developed.

VI. INTESTINAL DISTURBANCES DUE TO THE STOMACH, THE LIVER, AND THE PANCREAS.

Before we can understand the disturbances originating or inhering in the intestine itself, those disturbances which have their origin in the stomach and the large digestive glands must first be known. We will therefore take them up first.

A. Gastrogenic Intestinal Disorders.

The stomach as the cause of intestinal disturbance.—Frequent occurrence of connective tissue-remains in the feces.—Causes thereof.—Explanation of gastrogenic intestinal disturbances.—Therapeutics.

The view that a large part of the intestinal affections, especially the temporary catarrhal and functional disturbances, are only a consequence of foregoing gastric affections is extremely suggestive. In view of the close coöperation of both organs, it is very popular for the laity to ascribe a temporary diarrhœa to a "disordered stomach." It is a remarkable fact that this interconnection has for a time been altogether overlooked in medicine, and chiefly on the basis of *von Noorden's* investigations (41, 42), in which he proved that the digestion of food-stuffs could proceed normally in the case of patients with an increased or diminished or even completely lost secretion of the gastric glands, and that the metabolism of such patients did not necessarily undergo changes of any kind. Only recently has a change taken place: through *Einhorn* (43), *Oppler* (44), and various other investigators, it has been shown that there are diarrhœas and constipations of purely gastric origin, and that the former are generally a consequence of diminished gastric secretion, especially of achylia; the latter (the constipation), on the contrary, are a result of hyperacid conditions. This rule, however, has a few exceptions. *Schütz* (45), who has most recently investigated this subject, found extreme and protracted diarrhœas even in cases of only slightly changed acid secretion and recognized their cause, among other things, in atonic conditions of the stomach, that is in reduced motility. The particular patients presented the appearance of a severe serious disease.

That these observations are not isolated, but that gastrogenic intestinal disturbances are extremely frequent and therefore have a great practical significance, must be obvious to any one who has systematically employed my functional examination. *The appearance of microscopically recognizable remains of connective tissue in the stool after the test-diet is a simple as well as a reliable sign of this disturbance; it indicated that the stomach in the particular case is unable to perform the work which must be expected of it.* In my fecal investigations I myself have come upon undigested connected tissue remains so often that I do not hesitate *to declare the stomach to be most frequently the point of origin of the most varied intestinal disturbances.* I purposely say *the most varied,* for, as already pointed out above, remains of connective tissue are found not only in cases of diminished hydrochloric acid secretion—although undoubtedly they occur here most frequently, one might say constantly—but also under the opposite circumstances. Furthermore, in atony and in pronounced insufficiency of the stomach, even occasionally in trifling, recurring disturbances of the gastric activity, which cannot at all be explained by a simple investigation of the content of the stomach, but only through long-continued observation. How the failure to digest the connective tissue is to be explained in all these cases, is not yet determined. Whereas, it is at once intelligible in achylia, we must go back for our explanation in cases of hyperacidity to a deficient pepsin secretion, which we know can occasionally happen in spite of increased HCl secretion (31). Where the motility is reduced, processes of decomposition may be involved, etc. The work of the intestine, if we disregard the presence of undigested connective tissue, need not necessarily thereby be altered; the connective tissue can permeate feces that are otherwise normal, just like down. More often, however, the stool is not normal in other respects, being soft and mushy to a liquid (diarrhœa), or containing mucus and showing signs of decomposition. Similarly the results of stomach affections on the intestine are not uniform; they can vary from the slightest functional deviations to the severest catarrhs according to the duration of the condition, the sensibility of the intestine, the care which the patient gives himself, etc. When pronounced intestinal affections are present, the symptoms may become so prominent as to determine completely the appearance of the disease, while achylia, which is well known, frequently occurs without any disturbances on the part of the stomach. In the fact that the connective tissue test in these instances at once leads us on the right path, lies its value. It has, however, a greater advantage, in that it reveals the danger threatening the intestines when nothing else indicates it, when only now and then subjective symptoms or rapidly subsiding intestinal irritations appear, which, without this test, would be overlooked entirely.

The lientery of the connective tissue in stomach troubles has furthermore not merely a diagnostic significance. It also contributes very much to the *explanation of the origin of gastrogenic intestinal diseases,* and moreover gives us a *therapeutic indication* of value.

It was at first naturally believed that the cause of those intestinal disturbances was to be looked for in the absence of the antiseptic action of the hydrochloric acid in the stomach. This view rests on the investigations of *Kast* (46) and *Mester* (47), by whom it was duly established that in case of complete drying up of the hydrochloric acid secretion in the stomach, the amount of ethereal sulphates in the urine increases or —expressed more cautiously—may easily increase, but the converse conclusion from the ethereal sulphates in the urine to putrefaction in the intestine is not free from objection; and furthermore, *Mester* himself observed that in order to give rise to real putrefaction in the intestine, there had to be, in addition to the absence of the hydrochloric acid, as a further cause, an increased supply of putrefactive elements per os. Thus is explained, *e.g.*, why patients with achylia very easily get diarrhœa; for instance, when they have eaten an egg that is not very fresh, and why the regular taking of not too small doses of HCl at meals almost always improves the condition of these patients essentially, etc. But HCl is certainly not by itself the determining factor in preventing processes of decomposition in the intestine, as can already be seen from the fact that many patients with achylia (according to *Schütz* perhaps two-thirds) do not get intestinal disturbances; and besides, we see frequently strong decomposition in the stomach itself in spite of high HCl values. Our view of the origin of these fermentations of the stomach at present is to the effect that disturbances of the motor activity of the stomach are almost of greater significance than disturbances of the chemic processes; most serious, of course, is the result of the combination of both, as in pyloric cancer. *Schütz* correctly ascribes his cases of chronic diarrhœa along with atony, coexisting with sufficient acid secretion, to the passing over of special putrefactive elements from the stomach into the intestine. Indeed in some cases he even succeeded in showing some of these directly in the stomach and in the feces. We must therefore extend this casual relation somewhat further and say: *in cases of insufficient function of the stomach, fermentative and putrefactive elements can more easily establish themselves in the stomach, or pass it undestroyed and in this way produce secondary intestinal disturbance as a consequence.*

The *second point* which is to be considered for gastrogenic intestinal disturbances is furnished by lientery of the connective tissue. It demonstrates to us *ad oculos*, in a way which can be doubted by no one, that in cases of insufficient function of the intestine there is imposed on the intestine increased work which it is not always capable of performing. Raw or smoked connective tissue the intestine cannot digest at all; if it has once passed the pylorus undissolved, it is carried along as ballast until it is defecated. For the muscle fibers, the fat, and the starch it is of no essential importance whether the stomach digestion is perfect, for here the intestinal juices can enter and compensate. It is the connective tissue which especially becomes dangerous to the intestine. In order to be able to appreciate this fact we need but collect all the connective tissue which appears in the daily amount of the feces of one suffering from stomach trouble. By such experiments it can easily be shown, that under certain circumstances after the test-diet the connective tissue of the raw meat is quantitatively evacuated, so that it permeates the excrement as a mesh work and converts it into a down in form. It is necessary further to remember in this condition that smoked, raw, or half-raw (insufficiently cooked or roasted) meat taken in the free *choice of diet* is seldom chopped up so finely as the chopped beef in the test-diet. Raw ham is scarcely ever sufficiently minced. As a consequence of this in gastric disturbances not only isolated connective tissue is lost, but also the muscular and fatty tissue enclosed in it. It is normally the function of the gastric juice to prepare the pieces of meat and fat for disintegration through the dissolution of connective tissue—a function which it performs excellently, as every test-tube experiment shows. If this disintegration is absent, the pancreatic juice can only eat away the surface of these unreduced particles of meat, whereby, of course, much less is dissolved, and as a result pieces of meat and fat are evacuated (meat and fat lientery). The presupposition here is always that smoked or insufficiently cooked meat has been eaten, but this presupposition often holds only in case of persons with stomach disorders. Through Dr. *Brinck* (34) some years ago I had collected several cases of severe acute digestive disturbances in connection with severe intestinal colic, in which night-vessels full of undigested, putrefying bacon remains were evacuated at one time.

It is at once evident that the burden of undigestible matter, as it here confronts us, as a result of primary gastric disturbance, cannot be a matter of indifference to the intestine. *Schütz* presents the injuries occasioned thereby as an insufficiency of small intestinal digestion either gradual or sudden in its onset. The small intestine consequently, and above all the pancreas and the gall-bladder, can no longer comply with the increased demand made on them for so long a time, and the further consequences of this are diarrhœas and finally *intestinal catarrhs.* The condition seems to me to be even simpler: it is not at all necessary to assume as a connecting link a secondary insufficiency of small intestinal digestion, which moreover has not as yet been proved. Whether the small intestine functionate properly or not, it cannot digest the connective tissue, and the tissue elements enclosed in it at all. The mere passage of these through the small intestine would not injure it if they did not form a recess or breeding place for putrefactive matter which either at once or later causes irritation and subsequently inflammation of the intestinal wall. The inherent disinfectant power of the small intestine, which as *Schütz* (75) has shown, forms an essential support of the gastric disinfection, must remain ineffective against these putrefactive elements. This is confirmed by the beautiful results of *Strasburger* (7, 18), which prove that the amount of intestinal bacteria is dependent primarily on the quantity of food remains; the better the food has been digested, the fewer the intestinal bacteria. And so we must explain many cases of diarrhœa by the lodgment of fermentative and putrefactive elements in the insufficiently digested food-remains. Any one who demands too much of his digestive apparatus—in common parlance, stuffs himself—gets diarrhœa, not because he has incidentally swallowed putrefactive elements, but because he could not digest the quantity eaten, and thereby furnished the ubiquitous putrefactive microbes the oportunity to multiply and exert their power. The old saying, "Hunger is the best cook," has through *Strasburger's* investigations been restored to honor, for it has been shown that there is no intestinal antiseptic and no purgative which really diminishes the number of intestinal bacteria. That can only be accomplished through the removal of their nutrition.

I turn now to *therapeutic conclusions,* which may be inferred from the insufficient digestion of the connective tissue. They can be summed up in the requirement: *Remove half-raw and smoked meats from the diet in cases of gastric and intestinal disturbances!* This requirement, the basis of which I have discussed more in detail elsewhere (17), really follows of itself from the foregoing. We have seen that the intestines cannot digest raw connective tissue at all; it would therefore be foolish to offer it to a disordered intestine. We have seen further that an inability to digest connective tissue manifests itself very readily in stomach troubles, and, moreover, in stomach troubles of the most varied kinds, so that this fact forms a delicate test for this purpose. We would do well therefore to avoid raw meat altogether here, rather than to expose the patient with a disordered stomach to the danger of secondary intestinal disturbances. From this requirement we should deviate only when raw or smoked meat offers such corresponding essential advantages over meat cooked or well roasted that we are obliged to take the concomitant disadvantages along with it. What then is the situation?

To be sure, the view that raw meat is especially easy to digest is firmly rooted in the popular mind, and there are a great number of

physicians who think they can support this view on scientific grounds. The reasons which they adduce are the two following: that raw meat stimulates secretion and possesses a great affinity for acids. The first of these arguments has been proved by the well-known and interesting experiments on animals by *Pawlow* (32); it holds especially in cases of hypoacidity or anacidity of the stomach. But if we compare the advantages which can be secured for the HCl secretion by serving raw meat, with the disadvantages that are caused by the connective tissue which is introduced along with it, there cannot be any doubt but that the latter exceed considerably. This power of stimulating secretion has been proved only for the normally functioning stomach; in the case of a disordered stomach this power must certainly be very low. Moreover, it can be replaced in every case, as *Pawlow* himself has shown, by meat extracts, bouillons, solutions of *Liebig's* meat extract, etc. The second argument, the great affinity for entering into combination with acids, is eventually of importance for the treatment of hyperacidity. It contradicts to a certain extent the first-named argument; for the question may justly be asked: Of what use is the increased affinity for acid if at the same time there is an increase in the secretion of juices and *vice versâ*. In cases of hyperacidity the conditions are such that all therapy which proceeds from the symptomatic indication to combine the abnormally formed acid, must yield to the more important *indicatio causalis* of that therapy which seeks to restrain the *formation* of acid. The avoidance of irritation, not use or stimulation of the stomach is here the first prime requisite! I have, therefore, long ago banished altogether all food-stuffs that irritate or excite secretion from the therapy of hyperacidity, and I believe that I have benefited my patients more by this method than the so-called "albumin therapeutists" ("Eiweisstherapeuten"). If the acid must be neutralized, I prefer to administer some sodium, or to wash out the stomach, rather than to fill the stomach again and thus never to give it a rest.

We see therefore that both the arguments marshaled from the scientific standpoint in favor of raw meat, are not tenable. Only under one condition could raw meat be served, *i.e.*, if it could be freed from connective tissue. In practice this is as good as impossible, even if it is chopped up or scraped ever so fine. It is a delusion to believe that scraped up ham is free from connective tissue. We need only try to remove the connective tissue from ham or from raw meat as completely as the intestine does in case of achylia and it will be found this is impossible. The worst misunderstandings prevail in regard to smoked ham: smoked ham can never be ground up fine enough by the teeth, and yet there are people who do not consider it necessary to cut it up with the knife.

I will perhaps be confronted with practical instances and be told that we know a large number of patients whom we certainly could not have advanced in their nourishment without raw meat as much as we have succeeded in doing with it. It was the only thing they took in their severe sickness, etc. Perhaps, further, the example of the English will be adduced, who, the layman believes, eat all their meat half-raw. Both are false. Appetite is not the sole criterion of how food agrees, else mushrooms, crabs, and nuts would be digested easily; the examination of the feces is undoubtedly a more trustworthy standard. And as regards the English, they never think of eating their meat half-raw, as it is customary in North Germany; they eat it roasted rare, and that is quite a different thing. In meat roasted rare the connective tissue is dissolved, in the half-raw meat it is not. Therein lies the difference. In England raw meat is not prescribed for weak stomachs; that is only a German custom.

So much then for connective tissue lientery, on which I have already gone too far. To the chapter on gastrogenic digestive disturbances I will add only the practical advice that in all doubtful and protracted cases of diarrhœa, at least one experiment should be tried with HCl and a bitter, and wherever the aspect of the disorder is more severe, to additionally wash out the stomach. It will be a matter of astonishment to discover how often this therapy, which is scarcely ever thought of, is successful—a sign that the stomach is more often the starting point for intestinal lesions than is commonly supposed. These intestinal lesions consist chiefly of diarrhœas, and these I have had especially in view in the preceding discussion. The rarer constipations as conditions consequent on gastric disorders (especially in hyperacidity) will be again taken up by me later.

B. Hepatogenic Intestinal Disorders.

Significance of the secretory disturbances of the bile on digestion.—Influence of the bile on intestinal putrefaction and on peristalsis.

The hepatogenic intestinal disturbances are essentially conditioned by biliary obstruction. That also pleiocholia or pleiochromia of the bile (*e.g.*, in loss of blood) results in intestinal disturbances is not generally known. In England, at any rate, it is customary to ascribe certain indefinite digestive disturbances (variable coloration of the feces, pressure, and swelling in the region of the liver, yellow coloration of the conjunctiva and the skin, and a general malaise, etc.) to variations in the chloropoietic action of the liver (biliousness), but these symptoms are too indefinite to allow of a pathogenic explanation, and therefore this doctrine has never been able to become naturalized among us.

Biliary obstruction can be recognized usually by the white color of the feces, at any rate with certainty from the result of the sublimate test, but there is an exception, the so-called "acholic stool without icterus," which is yet an open question. *Nothnagel* (4), who first occupied himself with thorough investigation of these stools (where no *biliary obstruction* exists) formerly believed that they were to be explained by a temporary exhaustion of the biliary secretion, or by a functional hyposecretion. Later (22) he embraced the view of *v. Jaksch* (49) and *von Fleischer* (48), according to which the cause is either a deficient fat absorption in the *intestine* or the excessive reduction of bilirubin (to leucohydrobilirubin). In the latter case the feces on exposure to the air, take on a brown color on the surface. The sublimate test is here, too, the most reliable test; it never fails if hydrobilirubin or its reduction product at all is present, unless the stool is not subjected to examination early enough or has already undergone a putrefying decomposition in the intestines.

In general we can say therefore: Variations in the amount of fecal coloring matter and also in its chemic constitution, but without biliary obstruction, are not sufficient to cause a functional disturbance of intestinal digestion—deficient fat absorption. Biliary obstruction, on the other hand, always occasions imperfect digestion of fat; indeed, in the complete cessation of bile (absence of all fecal coloring in the sublimate test) 25 to 50 per cent. of the fat of the food reappears in the excrement undigested. This well-known and seemingly simple phenomenon is, however, by no means fully explained. How does it happen that the pancreatic juice is not alone capable of satisfactorily digesting the fat, especially since the stomach already splits the fat according to *Volhard's* (50) investigations. According to our knowledge of physiology, the effect of the bile on fat digestion is limited to the forming of an emulsion and the stimulation of the intestinal epithelia for absorption. We should therefore expect that after an emulsified fat (milk) is presented, a still larger portion should be absorbed. But this is not the case. We will not, however, enlarge upon these purely theoretical questions, but turn to a few *practical aspects* which are caused by the absence of the fat-digestion—only fat-digestion and not albumin and carbohydrate digestion is disturbed.

An old law still to be found in the more recent and even the latest text-books, says (in substance) that in the *absence of bile in the intestine, the putrefaction of ingesta is increased, and it leads to constipation. This law is false, for the very opposite is true.*

Whether the bile possesses such antiseptic qualities or not, the conditions in the intestine on which putrefaction of ingesta depends are quite different. Neither does the existence of an increased amount of ethereal sulphates in the urine determine the degree of intestinal putrefaction, as we will see later. On this point only the incubator test gives us information and, if need be, the weight of the fecal bacteria according to *Strasburger.* These two methods prove, however, the absence of decomposition: fatty stools do not ferment, not even if starch is added. Neither do they putrefy; fatty stools putrefy only as a result of pancreatic and intestinal affections in which the albumin remains are at the same time increased. They react at any rate strongly acid, but that is the result of the large content of higher fatty acids, and it is these fatty acids which spread the bad odor. We can perhaps speak of a decomposition of fat, but not of albumin putrefaction. In regard to the amount of bacteria in the feces *Strasburger* found in icterus a striking diminution. Hence, too, it is that icteric fatty stools scarcely ever cause catarrhs and inflammations of the intestinal mucous membrane. This is therefore of therapeutic importance, because from it we learn that the danger of occasioning intestinal disturbances through large amounts of fat in the food does not exist *de facto.* Fat is not at all serious for patients with intestinal troubles when it is served in suitable form.

Constipation in patients with cessation of the bile I have never as yet met. In uncomplicated cases of cessation of bile the stool evacuation proceeds without retardation; indeed, on account of its soft consistency and the fact that the amount of the feces is about three times as large as the normal quantity, the stool is, on the contrary, surprisingly thin and frequent. This observation *Boas* (51) has also made; it is probably to be explained in part by the slightly stimulative action of the free fatty acids, present in considerable quantities. This fact seems to me likewise to have practical significance in so much as it shows us a way by which it would perhaps be possible to overcome chronic habitual constipation. If we could succeed in increasing the amount of fat in the intestinal content to the point it reaches in icterus, of course without occasioning injury, then presumably the game is ours. But I will not spin out this thought further, it will again come in the discussion of constipation.

C. Pancreatogenic Intestinal Disorders.

Significance of the complete and incomplete absence of the pancreatic secretion for digestion.—Diagnosis of both conditions.—*Sahli's* method.—My method.—Therapeutics.

If the pancreas is completely extirpated in experiments on animals, or if it is completely destroyed in persons, there occur—aside from diabetes—the following disturbances: One-half of the albumin ingested is lost and similarly a large part[1] of the fat. In this connection it is a remarkable fact that here (in contrast to cessation of the bile) fat presented in emulsified form (milk) is better digested than unemulsified fat. With regard to the degree in which the fat is split, which according to the fundamental investigations of *Müller* (52) should be very much reduced, opinions are still at variance; the differences which have become known evidently come from the various kinds of ingested fats (emulsified or not), and perhaps from the varied intensity of the fat-splitting process in the stomach. The digestion of carbohydrates, as far as we at present know, is not hindered by the destruction of the pancreas; here it seems the intestinal juice steps in vicariously.

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[1] Writers here report very different amounts. Cf. (13).

In the partial destruction of the pancreas, which occurs more frequently in man than the complete destruction, no disturbance of any kind need appear in the intestinal function. The same is the case with impediments of secretion by the occlusion of the *ductus wirsungianus.* Just as pancreatic diabetes appears in experiments only after the last remnant of gland has been extirpated, so the disturbances in digestion as a rule then first become definite. *I say "as a rule," for there are exceptions.* But the fact that considerable constriction, softenings, and even apparently complete occlusion by a stone of the pancreatic duct can exist without disturbances of digestion necessarily following, is so striking a fact that it deserves to be especially emphasized. I regard it as superfluous to establish this fact with references in literature and cases of patients; I refer to the well-known monographs (53) and to the practical experience which constantly confirms it for us. More important is the question, how is this to be explained? Is the compensatory functioning of small gland remains indeed so great that they can for a considerable period take over the function of the entire organ? That is impossible; for we know that of another organ likewise important for nutrition —the intestine—a third to half can be removed without the subject necessarily suffering in his nutrition. With regard to the liver, also, it is known that its chief function, the formation of urea, is disturbed only when scarcely any of the functioning parenchyma is present. Or can perhaps the intestine step in vicariously? Even that would not be impossible, for we know from the latest investigations that *Brunner's* glands are connected genetically with the pancreas, and that the intestine is a synergist to the pancreas, in so far as the intestinal juice is necessary for the action of the pancreatic juice (54). Finally, for the cases of occlusion by stone, it may be supposed that the secretions reach the intestines in roundabout ways through the circulation; that the external secretion is, so to speak, converted into an internal secretion.

But be that as it may, we must meet this condition. *For practice we learn from it, that whereas the complete destruction of the pancreas can be recognized without any difficulty, the diagnosis of an incomplete lesion of it is attended with the greatest difficulties.* In the first case we have again—in so far as intestinal disturbances are concerned—first of all a fatty stool. This fatty stool is differentiated from the fatty stool in acholia by various characteristics.

1. There is no absence of biliary pigment,
2. the fat is often (not always!) present as neutral fat (in form of drops under the microscope),
3. it contains at the same time macroscopically recognizable muscle remains,
4. as a consequence of this complication, it sometimes shows putrefaction (either when fresh or after incubation).

On account of the putrefactive phenomena caused by the diminished digestion of albumin, the pancreatogenic digestive disturbances more readily give rise to secondary catarrhs than the hepatogenic, which, as already mentioned, can exist for months and even years without occasioning inflammation.

The diagnosis of a *partial lesion of the pancreas* can in individual cases be formed from the combination of symptoms here described, providing indications of them are present, but this is seldom the case. Recourse has therefore been had to special methods for diagnosis among which that of *Sahli* has obtained most recognition. *Sahli's* well-known method (38, 39) consists in treating gelatine capsules for a varying period of time with formaldehyde vapors, whereby they are made proof against the digestive effect of the juice of the stomach, but not against the more powerful pancreatic juice (glutoid capsules). The capsules are filled with iodoform. As soon as the iodoform is set free in the intestine by the dissolution of the capsule, it is absorbed, and this point of time shows itself in the iodine reaction of the saliva. The difficulty with which *Sahli* had to contend in this method and which, according to my view, it will never be possible to overcome completely, is to strike the "degree of hardness" for the capsules, suitable for all cases. For formalin has the property of making the tissue which has been impregnated with it proof against all digestion, tryptic as well as peptic. This can easily be proved by test-tube experiments. Since the degree in which pancreatic digestion is stronger than peptic digestion is by no means a constant one, but is subject to obviously large individual variations (according to the intensity of the secretion of the gastric and pancreatic juices; according to the length of time in the stomach, etc.), and, furthermore, since the preparation of capsules of a constant and uniform degree of hardness is scarcely possible, we cannot even from the start expect altogether uniform results even in the case of healthy persons. How much the less in the case of the sick with failure of the gastric juice secretion, etc.!

Practical experience has unfortunately confirmed these fears. Already from *Sahli's* own numerous observations and still more definitely, from those of *Fromme* (55) and those undertaken at my instance by *Wallenfang* (56), it appears that from a retarded glutoid capsule reaction the diagnosis of a disturbed function of the pancreas cannot always be made with certainty. On the other hand, the converse, that a prompt reaction excludes the possibility of a disturbance in the pancreas, seems justified. We can therefore attribute to *Sahli's* method only a negative diagnostic value, and that naturally reduces its value very considerably.

For this reason I have occupied myself for a considerable time with experiments, which aimed to supplant or supplement *Sahli's* method through a positive proof. These had as their basis my more careful study of the varied behavior of tissue elements in the gastric and pancreatic juices (30). This showed itself, among other things, in that the nuclei of the cells, in direct contrast to the connective tissue, which was discussed above, are digested only by pancreatic secretions, but not by gastric juice. If undigested tissue nuclei reappear in the feces, we may then safely conclude to an unsatisfactory functioning of the pancreas, just as similarly from the appearance of connective tissue we may conclude to insufficiency of gastric digestion. In order to be able to find the piece of tissue served again in the feces, it is best to give two small cubes of meat[1] which are placed in a little silk gauze bag in a wafer with the test-diet and repeat for several days at noon.

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[1] **Fresh (somewhat marbled) beef is cut in cubes of about $^1/_2$ to $^3/_4$ centimeter thick and preserved in alcohol. After hardening they are placed in very small gauze bags and again preserved in alcohol. Before using the little bags must be well washed in water for several hours.**

The little bags can easily be found again in rubbing up or sifting the excrement (through Boas's stool sieve), they almost always contain small remains of the tissue. These are then rinsed off in water and examined either fresh (with acetic acid or solution of methylene blue) or (in doubtful cases) after preparatory hardening and staining, for the presence of nuclei.

The experiments on dogs, which *Wallenfang* performed to test this method, have shown that, whereas no nuclei could be demonstrated in the meat-remains in the feces of the dog, after the complete extirpation of the pancreas (after the complete extirpation only and not after the partial!) the nuclei were preserved. From this method we may expect also practical results in man and the observations which I have thus far been able to collect seem to confirm this. Unfortunately these are not yet complete enough, *i.e.*, as regards the results of dissection, to permit a final judgment for the present. I can only say that, whereas the nuclei are never preserved, neither in disorders of the liver and intestine nor of the stomach, they are preserved in the rare cases of destruction of the pancreas. At the same time I will not fail to mention that this method does not yet promise to become an ideal one. For one thing, the demonstration of the nuclei is not always simple, deceptions through elements in the feces occur. Then, in intense diarrhœa single nuclei hidden in the remains of the preserved tissue occasionally remain intact even in the absence of disturbances of the pancreas. (It is best therefore to adhere to the rule that only the ***preservation of all the nuclei*** or the larger part of them allows inferences for diagnosis.) Finally, and this is most important, in cases where the feces remain in the intestines for more than thirty hours, intestinal putrefaction is of influence in determining the result. Putrefaction works in the same way as pancreatic juice, it allows the nuclei to disappear be it through necrosis or through solution. Here, too, therefore we cannot convert the proposition, we cannot infer from the absence of nuclei that the pancreatic secretion is intact, at least, if the little sack has remained in the digestive canal longer than twenty-four hours, or if strong intestinal putrefactive processes exist. If therefore we wish to be safe we can only avail ourselves of the positive result, this is unequivocal; it reads: *if all the nuclei reappear, there exists an extreme disturbance of the pancreatic secretion.*

Undoubtedly this positive test in connection with the negative test of *Sahli* will enable us to make a step forward in the diagnosis of the disturbances of the pancreatic secretions. But it does not allow us to attain the desired goal—to reveal slight disturbances of this kind. Slight disorders of secretion, as has been already repeatedly pointed out, are so completely compensated for, in ways which we do not yet know, that as a rule they do not leave behind obvious functional disturbances.

For practical therapeutic use the investigations thus far described are not yet available. Still it ought to be pointed out here that the organo-therapy of the pancreas, the administration of pankreon in certain or doubtful disturbances of the pancreatic juice is not only of therapeutic, but also of diagnostic value. Under certain circumstances, it enables us to make a differential diagnosis of intestinal disorders, especially of absorptive disorders, for the latter, it is supposed are not favorably affected through the administration of pankreon, while the former are [*Salomon* (28)].

VII. INDEPENDENT INTESTINAL DISTURBANCES.

The digestive disturbances inhering in the intestine itself are very numerous and manifold. An exhaustive presentation of them is naturally not intended here, as in the preceding only a few diagnostic, pathogenic, and therapeutic points are to be taken at random, especially such as have been obtained from the systematic application of the test-diet. The superscription: "Independent Intestinal Disturbances" is therefore chosen, because the usual classification into primary and secondary intestinal affections easily gives rise to false notions. The independent intestinal disorders do not by any means always originate in the intestine. As we have seen, a gastrogenic intestinal digestive disturbance can gradually assume an independent character, and as such it can continue to exist for a time even after the gastric disorder has terminated. Further, there are severe diarrhœas which must be regarded as localizations of general infections as in erysipelas, pneumonia, septicæmia, etc.

In the frequent processes of infection of the intestines (typhoid fever, tuberculosis, cholera, dysentery) the situation is probably such that the point of entrance of the disorder is to be sought in the intestinal mucous membrane itself; why the body lodgment of the exciting causes of the disorder takes place just here and how in individual cases the disorder arises, are questions about which we are not sufficiently informed. Presumably in this connection the stomach plays a part, for it is to be regarded as an established fact that in epidemics of typhoid fever and cholera, disturbances of gastric activity strongly predispose to infection.

1. Organic Disorders.

Relation of the organic to the functional disorders of the intestines.—Causes of diarrhœas in cases of ulcers.—Dietetic inferences.—Catarrhal and nervous forms of diarrhœa.—Diet in chronic catarrhs.—Salicylic milk.—Astringents.—Intestinal atrophy.

With regard to the independent disturbances of intestinal activity in so far as they will concern us here, stress is to be laid on the fact that they seldom run their course as purely functional disturbances, *i.e.*, without anatomic changes of the mucous membrane. We must therefore include ulcers and catarrhs within the sphere of our consideration. The relation of organic to functional disturbances, especially in the intestines, is an intimate one, but it is at the same time so very variable that for the most part a clear understanding of it is to be obtained only with difficulty As an example I take at random, first, the diarrhœas in intestinal tuberculosis and intestinal typhoid.

A. ULCERS.

It is almost universally known that diarrhœas form a frequent though by no means constant symptom of typhoid and intestinal tuberculosis and of intestinal ulcers in general. *Nothnagel* (4) in his fundamental investigations has duly appreciated this fact. There are cases where, in spite of the intense ulceration of the intestinal wall, entirely normal stool, and even constipation exists. On the contrary we occasionally see *intra vitam* severe uncontrollable diarrhœas, whereas, *post-mortem*, the intestine shows only a little isolated ulcer or only a cicatrix. Instances of the one as well as of the other kind I have investigated thoroughly *post-mortem* macroscopically and microscopically, and convinced myself that the mucous membrane is not destroyed by ulcer; and this is true even for the immediate neighborhood of the ulcer. A concomitant diarrhœa is often absent in cases of ulcers. If diarrhœa exists, it can only arise through the irritation of the base of the ulcers; and the varying degree of sensibility of the latter (or of the nerves exposed in it) explains completely the various clinical conditions. In other words, the diarrhœas in typhoid fever and intestinal tuberculosis are sometimes of a purely nervous nature. I lay stress on "sometimes," for in other cases we find the mucous membrane more or less inflamed, and then the accompanying catarrh often comes to the front in the clinical picture of the disorder. In the examination with the test-diet we then fail to find the little mucous flakes of golden yellow color, which, judging from their microscopic characteristics, come from the small intestine. These flakes of course can come only from the mucous membrane itself and not from the base of the ulcer; they give us a rather important indication not alone for the understanding of, but also for the treatment of, an individual case.

The treatment of intestinal ulcers, especially of the tubercular, is a very thankless undertaking. Previously, in the discussion of the test-meal, I have pointed out that of the cases in which milk is not tolerated by the patients, a large number are those of intestinal tuberculosis. No kind of dietetic regimen can here lay claim to general validity; indeed, we even see frequently that very coarse diet, especially vegetables, agrees excellently (at least for a time), whereas, every bland diet causes uncontrollable diarrhœa. That holds especially for the cases that run their course without inflammatory complication, even if the ulcers in such cases are ever so extensive. Naturally this does not hold for all cases, for, as has been said, the irritability of the base of the ulcer varies. Nevertheless, in practice, in cases of intestinal phthisis, where after the test-diet the signs of catarrhal inflammation of the mucous membrane (mucus, putrefaction) are wanting, or are insignificant in the excrement, we can always find justification from the above not to limit the diet too closely, but to vary the food prescribed, and occasionally even run some risk. In typhoid fever naturally we are more loath to do so; and yet I should like to express it as my personal conviction that in normal conditions of stool and in typhoid fever such an exclusively liquid diet as is now customary is no postulate. I myself would have no scruples against softened zwieback, *e.g.*, which is dissolved long before it reaches the intestinal ulcers, or against butter or broth made of potatoes and rice strained through a hair sieve, or even against finely chopped meat freed as far as possible from connective tissue. Relapses we find occur as often among patients who have been under the strictest care as among careless private patients.

B. CATARRHS.

As in intestinal ulcers the organic and nervous elements in varying relations governed the course of the disease, so also do they in the simple intestinal catarrhs. On other mucous membranes as in the respiratory tract, the stomach and the bladder, etc., we have likewise nervous symptoms complicated very generally by organic disturbances. The pertinently named "mucous colic" of *Nothnagel* resembles asthma so completely that both conditions have been compared in many respects with each other, and asthma is just the type of a nervous catarrh (57). The more cases of chronic diarrhœa I see, the more am I convinced of the fact that many cases passing under the diagnosis of nervous diarrhœas are slight chronic catarrhs, the varying clinical picture of which, the nervous elements influence largely. Most clearly the combination of catarrh and nervous irritation confronts us in *enteritis membranacea,* a process located in the large intestine exclusively. Now it is stimuli going out from the genitalia, now psychic influences, and again, sensitiveness to certain definite foods, or temperature, even to the movements or position of the body; which are associated with often altogether insignificant and circumscribed conditions of inflammation in the mucous membrane. Analogous processes are naturally in the small intestine. Here, too, in every case the exact functional examination will at once clear up the situation, inasmuch as the fecal examination will show either small mucous flakes or marked fecal putrefaction, which, as explained in detail above, is maintained chiefly through the admixture of inflammatory products from the mucous membrane. The excrement itself shows then no symptom of disturbance of function; that is, the meat is well digested.

The therapy of these processes must be instituted simultaneously at both ends, at the inflamed organ and at the nervous system. As one or the other component is predominant the greater stress is to be laid on one or the other. We wish to consider here only the local treatment and here I would further like to discuss briefly two points which I have learned as a result of the function test. The one concerns the diet, the other medicinal therapy. The diet in intestinal catarrhs should be as unirritating as possible, easily digestible and non-putrefying. To meet these three requirements at once is indeed hard: in the first place, the difficulty is to be approached with the old established gruels (schleimsuppen) presupposing that milk does not agree. It is necessary always to aim at accustoming the intestine to milk again, that food-stuff, which with high nutritive value claims relatively the smallest amount of digestive work—and this is best accomplished by adding gradually to the soups a constantly increasing quantity of milk. The reason why the gruels, especially the carbohydrate food-stuffs, are to be preferred in intestinal catarrhs is a very simple one, they do not putrefy. Wherever the carbohydrates and albumins are together subjected to decomposition, albumin putrefaction does not set in as long as there are present carbohydrates capable of fermentation; the carbohydrates to some extent shield the albumin (here the inflammatory product of the intestinal wall) from putrefaction. It is beyond doubt established that the milk diet furnishes the smallest amount of putrefactive products (58), and in that, too, it surpasses all other food-stuffs. Of course, we must reckon with the fact that with our diet, intestinal putrefaction suddenly passes over into intestinal fermentation which the incubator test then shows. Obviously the irritating effect of the milk is restricted also in this way. In itself this is not so bad, for fermentation is at any rate the lesser of the two evils, but we should try to prevent it, and the simplest and surest way of doing this is by the temporary addition of small amounts of salicylic acid to the milk (or also to the soups) in the manner which I have already discussed in the test-diet. As *Strasburger* has shown, next to the reduction of the amount of nutrition altogether—this is also the best method to reduce the number of intestinal bacteria. Purgatives and intestinal disinfectants, even calomel, fail completely in this point. On the basis of my experience heretofore I should like to express as my opinion that salicylized milk has stood the test excellently, not only with adults, but also with children, where we unfortunately have to deal too often with severe intestinal catarrhs. It removes the injurious qualities of the milk without impairing its good qualities.

As regards medicaments, outside of opium, astringents are to be considered especially in chronic catarrhs. In the oft-quoted work of *Strasburger* it has been proved that these remedies diminish the bacterial content of the feces better and with more certainty than purgatives. Calomel, which is still regarded as the intestinal disinfectant κὰτ' ἐξοχὴν (*par excellence*), even increases the fecal bacteria, as is now established beyond a doubt (7, 8, 75). What I wish to add in regard to astringents is only that those administered in insoluble form (as tannigen, tannalbin, powdered bismuth) may occasionally produce irritation on very sensitive persons. The inflamed intestine is extremely sensitive to all insoluble and undissolved substances (remains of cellulose, potatoes not divided fine enough, carmine powder, etc.). Systematic fecal examinations show this. Not only all the food substances should be given in soluble form, but also the medicines, and that is best achieved through the chemical combination of both. Therefore, I give the tannin, *e.g.*, in the form of (rather costly) "milk-somatose" (somatose of milk albumin with 5 per cent. of chemically combined tannin), a preparation with which I have had excellent results, even with very sensitive intestines (59).

C. INTESTINAL ATROPHY.

Here a few words more concerning the interconnection of long-continued intestinal catarrhs or putrefactive decomposition of the intestinal content with severe (pernicious) anemias may find a place. On the basis of clinic and anatomic investigations *Nothnagel* (4) years ago described the atrophy of the mucous membrane, *i.e.,* that of the ileocecal region, as a frequent result of chronic catarrh and assigned to it a special place in clinical pathology. At first these investigations aroused but little interest, but as the atrophy of the gastric mucous membrane came to be better known, they were again taken up and the now widespread view developed that the atrophy of the mucous membrane of the stomach alone as a rule did not involve severe disturbances of the general condition of health, but that if the process extended into the intestinal mucosa, general disorders and, particularly, pernicious anemia could easily result from it. This view which obviously is based on the entozoön anemia is, however, without any basis in fact. All the more recent, who have carefully studied the question of intestinal atrophy, and I myself am in entire accord with them (60), have had to convince themselves that there does not exist an anatomic process which expresses itself as an atrophy of the intestinal mucous membrane. The illustrations on the basis of which *Nothnagel* founded his belief are obviously for the most part works of art, and *Nothnagel* (23), with the unselfishness of the true scholar, has admitted in the last edition of his "Erkrankungen des Darmes und des Peritoneums" (Diseases of the Intestine and Peritoneum), that he might have been mistaken. Also, the clinical fecal conditions, which *Nothnagel* described as characteristic of intestinal atrophy—an increased content of water with the absence of any other anomaly—scarcely occurs when investigated with the test-diet; almost always where there is an increased content of water, either products of inflammation (mucus) or indications of a functional disturbance (undigested food-remains) are present at the same time. But in spite of this the theory of the enterogenic origin of pernicious anemia has persisted; it has merely assumed another form. *Grawitz* (61), who has further defended it, supposes that, in absence of atrophy, it is also certainly possible that in cases of protracted catarrhs, abnormal products of decomposition of the intestinal content should exert a toxic influence on the blood tissue (Blutgewebe). How many pernicious effects have been attributed to the abnormal products of the decomposition of the intestinal contents, and how little positive evidence has remained after the critical sifting by *Fr. Müller* and *Brieger* (62) of the autointoxications arising in the intestine.

If the patients who suffer with pernicious anemia are systematically examined by means of the stomach tube and the test-diet, it certainly appears that the great majority of these suffer from achylia gastrica and the disorders of intestinal activity consequent upon it (catarrhs and increased putrefaction in the incubator test). Exceptions occur, however, as *Einhorn* (63) especially has lately emphasized. But how many patients are there suffering with gastrogenic intestinal disorders, the condition of whose blood is altogether normal. I estimate their number to be so high, that in comparison the little group of pernicious anemias disappears altogether. Already this condition of affairs speaks against a causal connection. And even more so does the chemical investigation of the products of putrefaction formed in the intestine which are found in the excreta of such patients. Hitherto for the purpose of orientation of the amount of enterogenic products of putrefaction we had at our disposal only methods of demonstrating these in the urine. *Strauss* (64), who carefully investigated a pertinent case by means of these methods, could not convince himself of an excessive intestinal putrefaction. Through *Ehrlich's* aldehyde reaction (65) we have now been put in a position to be able to estimate the amount of indol in the feces. *Baumstark* (66) has studied this reagent under my direction. He found in pernicious anemia an increased total amount of indol (feces plus urine), but throughout the amount varied within the limits, which occur in other cases of achylia gastrica. There does not therefore exist the slightest cause for the belief in an especially intense intestinal putrefaction or a specific putrefactive process as a basis of pernicious anemia.

2. Functional Disorders.

In spite of the intimate connection between organic and nervous affections of the intestine, which has been emphasized repeatedly in the preceding section, we can nevertheless mark off here a considerable number of different disorders of intestinal activity, which are exclusively or more cautiously expressed—quite predominantly of a functional nature, and which we may therefore designate in accordance with the principle *a potiori fit denominatio* as functional disturbances of the intestine. With the stomach, the heart, and sexual organs, the intestine is a favored organ for the localization of general nervous conditions. To the layman his opinion of intestinal diseases in general is the kind of evacuation—diarrhœa or constipation—which is regarded as the most important sign, in accordance with which the entire disease is to be gauged. From the physician's standpoint this conception is altogether fallacious. One diarrhœa is often entirely different from another diarrhœa, The conception of diarrhœa is of a too thin consistency of the stool and a too frequent evacuation. Both elements are indeed usually combined with each other, but to a certain extent they may also appear independently of each other. Thus there occur just in nervous conditions very frequent dejections without essential modifications of the feces, and again, on the other hand, one daily evacuation of a thin stool. Diarrhœa as a symptom is altogether too ambiguous for us to be able to derive from it anything more than a suggestion for further investigation. At any rate it is necessary always to bear in mind that in proving the presence of a diarrhœa—and the same holds with regard to constipation—a diagnosis has not yet been made. This can only be obtained on the basis of all the symptoms that present themselves and of a more protracted observation if need be.

Even if we must admit that the motor function of the intestine in one way or another participates in most purely functional disorders, the systematic application of the test-diet nevertheless shows us not infrequently disorders of intestinal activity which must be regarded as purely or almost purely secretory or absorptive.

A. SECRETORY DISTURBANCES.

Intestinal Fermentative Dyspepsia.—Jejunal Diarrhœa.

Among the former, the independent *secretory* disturbances, I would now count *intestinal fermentative dyspepsia,*[1] recognized by *Strasburger* and me through our function-test and described by us as a special form of disease. We were able to collect a rather large number of cases, where essentially subjective disorders existed: abdominal pains, borborygmus, diminished appetite, general malaise, whereas, on objective examination the patients gave the impression of being in tolerably good health. Now and then the abdomen was somewhat swollen, perhaps diffusely tender or tender only in the umbilical region, the tongue was somewhat coated, defecation ensued one to three times every day without real diarrhœa.

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[1] In our detailed communication concerning this (19) we left the question, what nature is the disease, still open; yet even at that time our thoughts inclined toward a functional disturbance, essentially secretory of the digestion of the small intestine. Further observations have strengthened me in this view.

Careful examination of the feces proved that the usually mushy stool was filled with numerous gas bubbles, it was foamy, light yellow in color, showed an acid reaction, and had a very strong odor of butyric acid. In other cases these characteristics were wanting, the stool appeared normal on macroscopic investigation. In the incubation test there always appeared, however, pronounced CH (carbohydrate) fermentation. There must have been involved a deteriorated digestion of starch, and only this, for other signs of abnormal function (muscle-remains, etc.) were completely absent. Neither did the examination of the content of the stomach show anything pathologic. The chemic analyses which we performed in eight cases also confirmed this assumption; we did not find the quantity of N (nitrogen) and fat in the feces greater than in normal stools, but, on the other hand, we found the CH content almost doubled.

If we consider that in these cases the stomach was healthy and that the pancreas and the bile must also have had their normal function, nothing remained but to attribute the cause for their aspect of the disease to a disorder in the secretion of the succus entericus. Of the succus entericus we know that it can develop powerful amylolytic actions and that these eventually extend into the large intestine. Certainly the disorder in secretion here in the foreground, in so far as the motility participates at all (somewhat increased evacuation), is obviously only secondarily drawn in sympathetically through the acid character of the intestinal content, for the absorption is altogether undisturbed. Clinically viewed, the affection proved in general a very slight one; for the most part it was easily removed within a short time through rest in bed and a suitable diet, sometimes in combination with warm applications and the administration of tannin or bismuth preparations. A few cases, however, remained intractable.

To what extent the *jejunal diarrhœa* of *Nothnagel* (22) is connected with this intestinal fermentative dyspepsia cannot for the present be decided. *Nothnagel* describes under it conditions of diarrhœa in which the content of the small intestine *quasi* in the same condition in which it exists in the upper part of the small intestine is evacuated from the body as an inodorous acid mass, very much intermixed with unaltered bile, and therefore a thick, tough, slimy mass, in which unaltered particles of food are abundantly present, especially remains of meat and of starch. Several times I have observed these conditions, which in themselves are rare, but have not yet had any opportunity to enter more in detail on them. So much, however, seems at once clear to me, that here the motor function of the intestine plays a large part, and that, if a disturbance of secretion is involved here, it cannot be restricted to the intestinal juice alone, but must include also the pancreas.

B. ABSORPTIVE DISTURBANCES.

Organic and Functional Disorders of Absorption.—Disturbances of gas-absorption.

While almost all organic and also most functional diseases of the intestine, namely, increased peristalsis, secondarily injure absorption, isolated independent absorptive disorders of the intestine are very rare occurrences; indeed, we must occupy ourselves very thoroughly with these matters if we would find them. As typical examples of disorders of absorption are the digestive disorders associated with diarrhœa, which appear as a consequence of congestions of the intestinal mucosa (weakness of the heart, cirrhosis of the liver), of amyloid degeneration of the intestine and of diseases of the mesenteric glands *(tabes mesaraica)*. But in all these conditions the picture is seldom a simple one; conditions of inflammation or ulcerative decomposition of the intestinal mucosa are scarcely ever absent. There are, however, observations of this kind (67) where these objections do not hold, and these prove that when there is exclusively or predominantly a disturbance of absorption, the absorption of fat suffers chiefly, the absorption of albumin less, and the digestion of carbohydrates least, if at all. The amounts of food that are lost are by no means very great, being far behind the loss observed in affections of the pancreas. Nevertheless the differentiation is certainly not easy in doubtful cases.

Alongside of the organic disorders of absorption, which were cited here only for the sake of comparison, there are also *purely functional* ones, only that they are not as prominent, and therefore easily overlooked. It is indeed self-evident from the start that the amount of absorption shows variations among different individuals as well as at different times in the same individual, perhaps in the same way as the amount of the daily food ingested, the urinary excretion, etc. As a matter of fact, even with a diet as uniformly composed as our normal diet, the systematic investigation of the feces of healthy persons if extended over greater periods of time does not at all give the same result constantly, but there is found occasionally a little more fat-remains than usual, or other deviations from the condition of the normal excrement, which prove the occurrence of such variations. Even more so is this the case in many patients with indefinite digestive disturbances, irregular stool evacuation or other disorders pointing to the intestine, which, just like many intestinal catarrhs, as a rule pass from physician to physician under the diagnosis "nervous intestinal trouble." Thus I have often seen that such patients regularly excrete surprisingly many and large microscopic remains of muscle fibers, while in other respects the conditions of the stool are normal; at other times only the absorption of fat was disturbed. To the latter category of cases *Salomon* (28) has made an interesting contribution at the last Kongress für innere Medizin. He was able to prove that the disorder could not be ameliorated by administering pankreon (in contrast to the disorders of fat absorption in pancreatic disturbances), and he justly saw in this a valuable aid for differential diagnosis. Evidently there belong here also many of those "acholic fatty stools without icterus." But I will not go further into these intricacies of diagnosis. Therapeutically these cases are naturally especially welcome, because they can easily be ameliorated by the diminution of that part of the food-stuff which especially is not well absorbed.

There is yet to be mentioned here briefly a special kind of absorptive disorder, which undoubtedly occurs as a seemingly independent anomaly of a diminished absorption of gas on part of the intestinal wall, the consequences of which (flatulence and tympanites) are mostly very unpleasant for the particular patients. For the normally functioning intestine it may be taken for granted that by far the largest part of all the gas present in it is removed, not through the anus, but by way of the blood through absorption. How else would the fact be intelligible, that persons in good health can take without any trouble colossal amounts of drinks containing carbonic acid? It is self-evident that flatulence and tympanites can have their basis also in increased gas formation or diminished peristaltic motion, but these factors can easily be excluded with certainty. As far as my experience goes I must say at any rate that also in the other cases that still remain, a careful investigation of the excrement almost always reveals indications of a catarrh still existing or coming to an end. So it is also in the stomach. Any one who complains of distention in the region of the stomach, eructation, and the frequently accompanied pyrosis, is generally suffering from an "injured stomach."

C. Motor Disturbances.

Among the various affections generally described as disorders of the motor function of the intestine, there are relatively few which occur as independent disorders; the majority have significance only as the symptom of a disease, an accompanying phenomenon of other local or general disorders. This holds good, *e.g.*, for all cases of paralysis of the intestine and intestinal colic, and also for the well-known lead colic, which, as *Nothnagel* (22) justly emphasizes, is more a neurosis of sensation (a neuralgia of the plexus mesentericus) than a condition of spasm. Somewhat independent disorders are really only the peristaltic unrest of *Kussmaul (Tormina intestinorum nervosa),* the nervous diarrhœa and the form of chronic constipation that is based on intestinal atony.

Tormina Intestinorum.

I may presuppose that the diagnosis of *tormina intestinorum* of the rotatory movements of the intestinal coils accompanied by constant rumbling and splashing, with the ultimately visible peristalsis is well known, but I might, nevertheless, point out again that only when the other investigations, especially those of the stomach and of the feces, have given negative results is it justifiable to regard it as a neurosis. Taking the test-diet as a standard, this occurs but seldom; still it may be that my experience on this point is still too limited. In the more careful investitions of peristaltic unrest there is felt especially the want of a method which would enable us to measure the length of time which foods remain in the small intestine. For it is beyond a doubt just here that the small intestine is either exclusively or especially the part affected; the stool evacuation ensues throughout normally and regularly.

Nervous Diarrhœa.

The question of *nervous diarrhœa* is yet far from settled. That is evident from the fact that it is treated by *Nothnagel* and *Ewald* under the neuroses of motility, by *Boas*, on the other hand, under the neuroses of secretion. The existence of a purely nervous diarrhœa, that runs its course without any anatomical lesion of the intestine, is beyond a doubt. It is sufficient to refer to the constantly cited example of the diarrhœa produced by fear. Further, every physician has probably seen at times patients in whom the thought alone that they would have no opportunity for defecation soon suffices to call forth a movement, or in whom very definite psychic causes lead to evacuation. Moreover, in most of the cases that belong here the proof that anatomic changes in or on the intestine (cicatrices, catarrhs, adhesions) are absent can hardly be urged; and basing on the results of functional diagnosis, I have already expressed my conviction repeatedly above, to the effect that on careful investigation of the feces the host of nervous diarrhœas will shrink to a very small group. Besides, referring to the particles of mucus occasionally found, I have in this connection cited the accompanying abnormal processes of fermentation and putrefaction, which here are but seldom absent; whereas, they do not fit in with the clinical picture of nervous diarrhœa.

I must, however, remark here, that only the result of the investigation of fresh excrement is to be considered, not that of the incubator test. If the diarrhœa is really of purely nervous origin, the freshly evacuated excrement ought not show any signs of pronounced putrefaction or fermentation, it must have exactly the same appearance as the fresh content of the intestine. On the contrary, however, since it may naturally include abundant unabsorbed remains of starch and albumin, it may readily ferment or putrefy in the incubator test. Usually it also contains dissolved albumin, and on account of these varied admixtures —disregarding the processes of decomposition—it is scarcely possible to mark off this ailment from neuroses of secretion and absorption.

If we add to this, that if a person with normal digestion suddenly has, as a result of fright, thin stools, this is scarcely to be explained without assuming that there also takes place at the same time a serous transudation on the part of the intestinal wall, and that, on the other hand, there are cases where the emaciation is disproportionately large in comparison with the intensity of the diarrhœa; where therefore the absorption is obviously involved, we come to the conclusion that a nervous diarrhœa, as a consequence of isolated motor disturbance, scarcely exists at all; that here we are probably always dealing with a combination of various functional disturbances.

But even if the stool investigation must here often be inferior to the other clinical signs for the diagnosis of the condition, nevertheless it gives us a valuable suggestion for the treatment; I mean especially the dietetic treatment, for the total treatment must be preëminently physical or general. When this or that food-stuff is especially poorly digested, the composition of the diet must in such cases be accordingly altered; I would, however, recommend very particularly that the trend of the processes of decomposition, either in the fresh feces, or in that kept in the incubator, be taken as the starting point in the choice of diet. *In fermentation a predominantly albuminous diet, in putrefaction a carbohydrate diet, is the rule.* A sudden change of diet is occasionally of surprising efficacy in its results, even, *e.g.*, in the form of a vegetarian regimen. The scientific basis of this procedure is explained by the fact that the flora of the intestinal content requires some time in the case of a sudden change of diet before it can adapt itself to the culture medium [*Lembke* (68)]. During this intermission the irritations of the products of decomposition are lost, and that is of great advantage. Another thing I would always advise—rest in bed. It is beyond a doubt that with individual patients every bodily agitation, even that connected with spontaneous movement, suffices to relax peristalsis, just as, conversely, with others travel on the railroad or on a steamship unfailingly occasions constipation. Partly to the rest in bed connected with the functional examination, which I have always practiced at least for a few days; partly to the fact that most patients were unaccustomed to the composition of the test-diet, I attribute some successful results, which at the start were never intended. Most vividly shall I remember, among others, a young man who had from earliest childhood suffered from diarrhœa, which had defied all treatment. I explained to him that I would pass judgment on his condition only after investigation by means of the test-diet, and to our greatest surprise, thereupon a normal stool ensued every other day.

Chronic Constipation.

Gastrogenic constipation.—Nature of chronic constipation.—Therapeutics.

Chronic constipation, to which we turn at the conclusion of our discussion, is a chapter so much discussed that it seems almost impossible to contribute anything new to it. It is self-evident that we are concerned only with the nervous form of it, as it is generally assumed, resting on atonic conditions of the musculature of the large intestine or abnormal conditions of its innervation, we may calmly confess, that in practice, under this standard—just as in the case of nervous diarrhœas—many an instance of secondary constipation in connection with chronic catarrh of the large intestine, and often with stomach affections, is included under this caption.

As regards the latter, *gastrogenic constipations,* it has already been emphasized, that hyperacidity very frequently accompanies constipation, while in achylia just the opposite, diarrhœa, is the order of the day. This is, however, not constant. Thus I have repeatedly had under treatment patients with chronic constipation, who at the same time suffered from achylia, in whom obviously the condition had developed gradually from a chronic gastrogenic intestinal catarrh. After systematic HCl treatment a rapid improvement ensued. Similarly, constipation with hyperacidity must naturally be treated from the stomach. Constipation with hyperacidity is moreover theoretically interesting, inasmuch as it suggests that the strong disinfectant influence of the increased HCl of the stomach is perhaps able to be so prejudicial to the growth of intestinal bacteria to the extent that the natural irritations of the peristalsis which proceed from the products of decomposition disappear. This would be the very counterpart of the explanation given above of diarrhœas after achylia. Whatever be the facts of the case, it is not yet safe to say. *Strasburger* found by means of his method of weighing fecal bacteria in a case that belongs here, strikingly few bacteria; but since the primary constipation regularly goes along with a diminution of the amount of bacteria, the question is hardly to be decided by this method. In general it must not be forgotten that more protracted constipation can have on its side, as a consequence, disturbances of the stomach, at least subjective, and that therefore we must not always look at once to the stomach as the starting point. *Ebstein,* (69) who has given special attention to the phenomena of the stomach in constipation, points out that in the cases of chronic constipation examined by him the HCl content and the digestive power of the stomach were normal.

If patients with chronic constipation are put on the test-diet, it is observed on examination of the stool that not only the outer form of the feces and its content of water are changed, but that also various other deviations from the normal excrement exist. In the first place, the total amount (marked off for three days) is strikingly small, and not only of the moist, but also of the dry excrement, freed from water. While in healthy persons with the test-diet an average within three days of about 60 Gm. of dry excrement is formed, we find in chronic constipation amounts of 46, 37, and even 30.8 Gm. It cannot therefore be only a question of simple thickening of the excrement through an increased absorption of water. Neither is the matter explained by a reduced content of bacteria, for even after the deduction of these the total amount of excrement is much diminished as compared with the conditions of normal excrement. How does this happen?

If the microscope is brought in, it strikes us that only excessively small remnants of food-substances can be seen. Here and there a miserable remnant of muscle fiber, no potato cells at all, few soaps of lime, almost everything is detritus. The chemic investigation confirms this condition: the nitrogen content as well as the fat and even the cellulose content, are considerably diminished. *We have therefore a better digestion of the food substance, a too good digestion!* This result, to which, I believe great theoretic importance is to be attributed, seems to me now placed beyond all doubt.[1]

The idea that perhaps an increased decomposition of the remains of the food-stuffs in the intestine gives the appearance of better digestion, must be dropped. For there can be in reality no question of an increased intestinal putrefaction or intestinal fermentation in constipation; this assumption continues to wander from one generation to the next like a fairy-tale. Indeed we often find in the urine an increase of indican or of ethereal sulphates, but with that alone little can be done. Examination of the excrement shows, on the contrary, the absence of all fermentation and putrefaction, even in the incubator test. To the absence of products of decomposition, gases, volatile fatty acids in the intestines, etc., alone is the insufficient work of the intestinal musculature to be traced. And that is in full accord with the small numbers of bacteria in the feces.

The connection of things in chronic constipation is therefore this, that in the first place the digestion is too good. Therefore there is furnished a culture medium too poor for the development of bacteria, and thereby a diminution of the number of intestinal bacteria, diminished products of putrefaction and disappearance of the natural stimuli of peristalsis. Whether all cases of constipation are thus to be explained may temporarily be left unsettled; at any rate, it is no small percentage.

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[1] The first allusion to these conditions is found in *Strasburger* (7). The results here repeated come from the investigation of my assistant, Dr. *Lohrisch*, which are to appear in detail in D. A. f. kl. Med. (Has since been published in Deutsch. Arch. f. klin. Med., 79, page 383, 1904.)

The question arises, whether this new information, which we owe to the function-test, is able to produce fruitful results therapeutically. I believe it is. *Roos* (70), and, later, *Strasburger* (17), have performed experiments through skillful introduction of cultures of yeast and bacillus coli communis to heighten the processes of decomposition in the intestine and thereby to improve peristalsis. The experiments failed utterly, *Strasburger* could not even produce thereby an increase of intestinal bacteria. That is natural, for if in reality the better digestion is the primary, and the degree of bacterial settlement is only a function of the remains of the food-stuffs (which are to be disposed of), the lever must be put to the digestion itself, the attempt must be made to deteriorate it, in order that the processes of decomposition may take place in a wider scope. In fact, for a long time we are working more or less unconsciously in this direction. If we prescribe for a constipated person a coarse diet containing abundant waste matter, *e.g.*, black bread, salads, vegetables, fruit, nuts, etc., we introduce the cellulose, not because as an undigestible substance it mechanically stimulates the intestinal wall, but because it protects the enclosed starch kernels and albumin substances from absorption and thereby gives the decomposing bacteria the opportunity to develop and form the splitting-products (the volatile fatty acids, the gases, etc.) which stimulate the intestine. All food substances that are rich in waste are less digested than foods free from waste, and it is easy with normal digestion, through the addition of potatoes or black bread, to cause fermentation of the feces. That is what we aim at in the case of constipated persons; but unfortunately Nature here often proves more powerful than our skill, the digestion of constipated persons is so good that it disposes of what even seems to be indigestible things. It is very surprising how little of cellulose remains is found again in macroscopic investigation of the feces of constipated persons who have taken a diet containing much waste. Chemic examination also confirms the fact that the intestine of patients with chronic constipation is better fitted to a large degree to dissolve, if not to digest, cellulose. (Normally, the intestine, as is well known, possesses this capacity only to a very limited extent.) Special investigations on the causes of these phenomena are still wanting, and we shall, therefore, content ourselves for the present with affirming the fact.

It is remarkable that in the effort to deteriorate the digestion of food in constipated persons, hitherto the lever has been put only to the carbohydrate components of the food. Why not try it with the fat? The fat is particularly suited for this purpose, because it does not, like albumin, give rise to intestinal putrefaction; further, because, as we have seen in stools, fatty on account of the absence of bile, it is possible, through the mildly stimulating effect of the fatty acids that are split off, and in consequence of the soft consistency which it gives to the feces, to produce a thin stool. The only presupposition involved is that we succeed in making the content of the intestine richer in fat, and, indeed, into the deepest parts of the feces. Therapeutically, we would suppose that this would be easy to accomplish, for the limits of assimilation for fat are much lower than, *e.g.*, those for meat; in vigorous adults it amounts, according to *Rubner* (71), to about 350 Gm. *pro die.* This number, which taken absolutely, is still high, and holds only for butter, acknowledgedly the easiest fat to digest. For fat that melts at a higher temperature (mutton fat, beef fat), and especially for such fat as is enclosed in connective tissue, bacon, for example, it is considerably lower. What aspect then does the matter take on in practice? I have for a long time made experiments in this direction and might designate my general opinion, in so far as it can be determined up to the present, as favorable. One of the difficulties of the increased fat intake is corpulency, which very often is combined with constipation. Such patients would grow still stouter, and that would not do. Difficult, too, is the selection of a suitable fat, especially of such a kind which has not too high a melting point (in order not to make the excrement too solid), and which is not absorbed as well as, *e.g.*, butter. Bacon, which is well adapted for the purpose, can be taken only in small quantities. Nevertheless, with large amounts of butter some very beautiful results can be obtained, and I would not omit to refer at this time to the recommendation by *Dörffler* (72) of butter for the cure of constipation in infants, which has found general approval in practice, and which may serve us as a guide in this respect. Also, the justly relied upon "oil clysters" of *Kussmaul* are to be mentioned here; they operate, it seems, in a way altogether similar to the increased fat in the food, in so far as through splitting off of fatty acids they develop a gently stimulating effect on the wall of the large intestine. This explanation is at least more plausible than the highly naïve idea already righteously attacked by *Ewald* (73) that the oil dissolved or softened the scybala. For this purpose it does not avail; at most it may contribute to better evacuation by making the mucous membrane slippery.

Two more points in the treatment of constipation may be discussed briefly. The one concerns purgatives. I will not enter into detail upon them, but will bring to your consideration only the following. If, as we may with certainty assume, the products of decomposition of the content of the intestine, particularly the volatile (acetic acid, lactic acid), and the higher fatty acids, just as the gases represent an important normal stimulus for peristalsis, it suggests itself to introduce them skillfully just where they are absent, or are formed in too small amount, before attempting the sharper stimuli—the real purgatives. From above we accomplish little by administering them; it is necessary to introduce them through the anus, in order that they may reach the spot where they can develop their effect as directly as possible. Practically, for this treatment only, the gases come in question, and among them, in the first place, CO_2. Following this line of thought, I have for some time made experiments with CO_2 or Seltzer-water clysters in constipation, and have had occasion already to recommend these to various colleagues with whom I came in contact in my practice. The application is very simple. On a Seltzer-water bottle, a stopper with a hole through it is placed, to which an intestinal tube is joined. Insert the latter and shake the inverted bottle until the CO_2 that develops has driven all the Seltzer water into the rectum. On the results of this treatment, for the present, I can speak only with reserve. The Seltzer-water clysters are not to be regarded as purgative clysters, or as a direct aperient; on the other hand, they seem, on prolonged application, to exert a refreshing, tonic influence on the mucous membrane. They cause hardly any discomfort; it is remarkable how much gas can be introduced from this end. For sensitive patients I have substituted oil of sesame clysters containing

CO_2 by means of the very useful "effervescent oil," introduced into the market by the firm *Dietrich-Helfenberg.* It seems almost as if the effect of these effervescent oil clysters were even safer and more effectual than injections of Seltzer water.

The second point is concerned with rest in bed as an aid in the treatment of constipation. Generally it is said that the constipated persons should move about very much, but there are many people who, in spite of very much activity in their calling, display a high degree of constipation. Indeed, at least passive motion in many respects tends to delay stool (riding, traveling on trains). Under such circumstances rest in bed in connection with a diet rich in fat (test-diet with the addition of milk or butter) often shows remarkable results. I do not therefore neglect to have the patients confined to bed during the function-test, which in this respect I used at the same time as a time for observation. Moreover, I am not alone in my experience. *Boas* (51) also cites cases of this kind. Obviously, whereas, the agitation of the intestines through walking stimulates peristalsis in those disposed to diarrhœa, in others it serves as an inhibitory stimulus. As long as we have not entered deeper into the complicated condition of the innervation of the musculature of the large intestine, we must unfortunately, rest content with such vague explanations.

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